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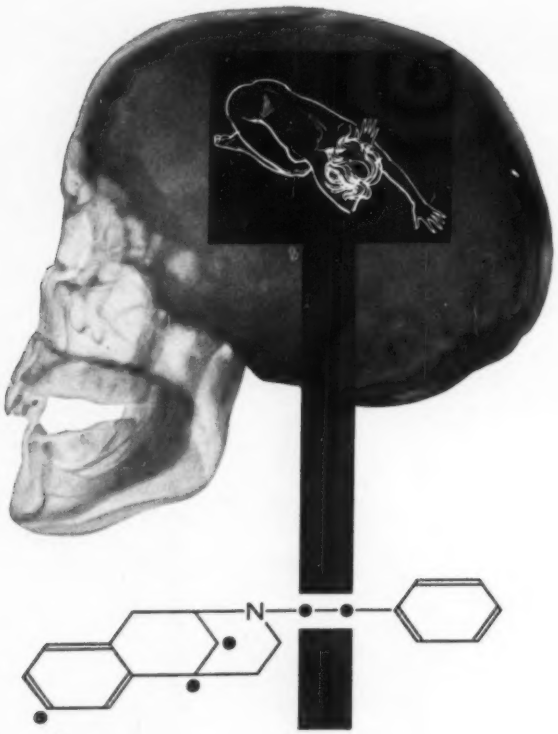

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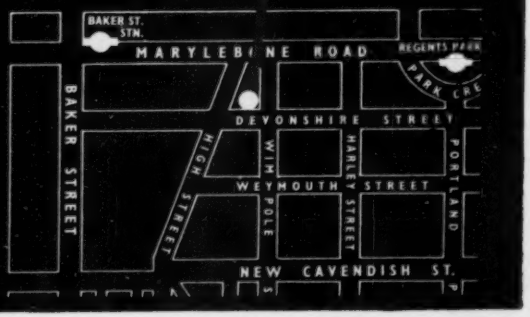

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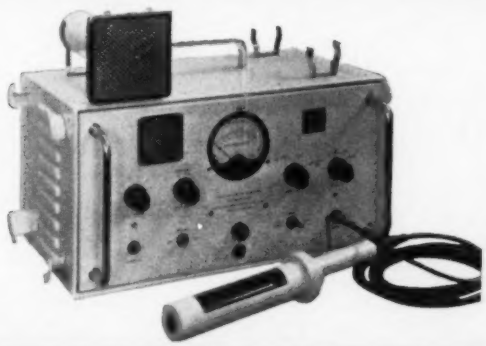

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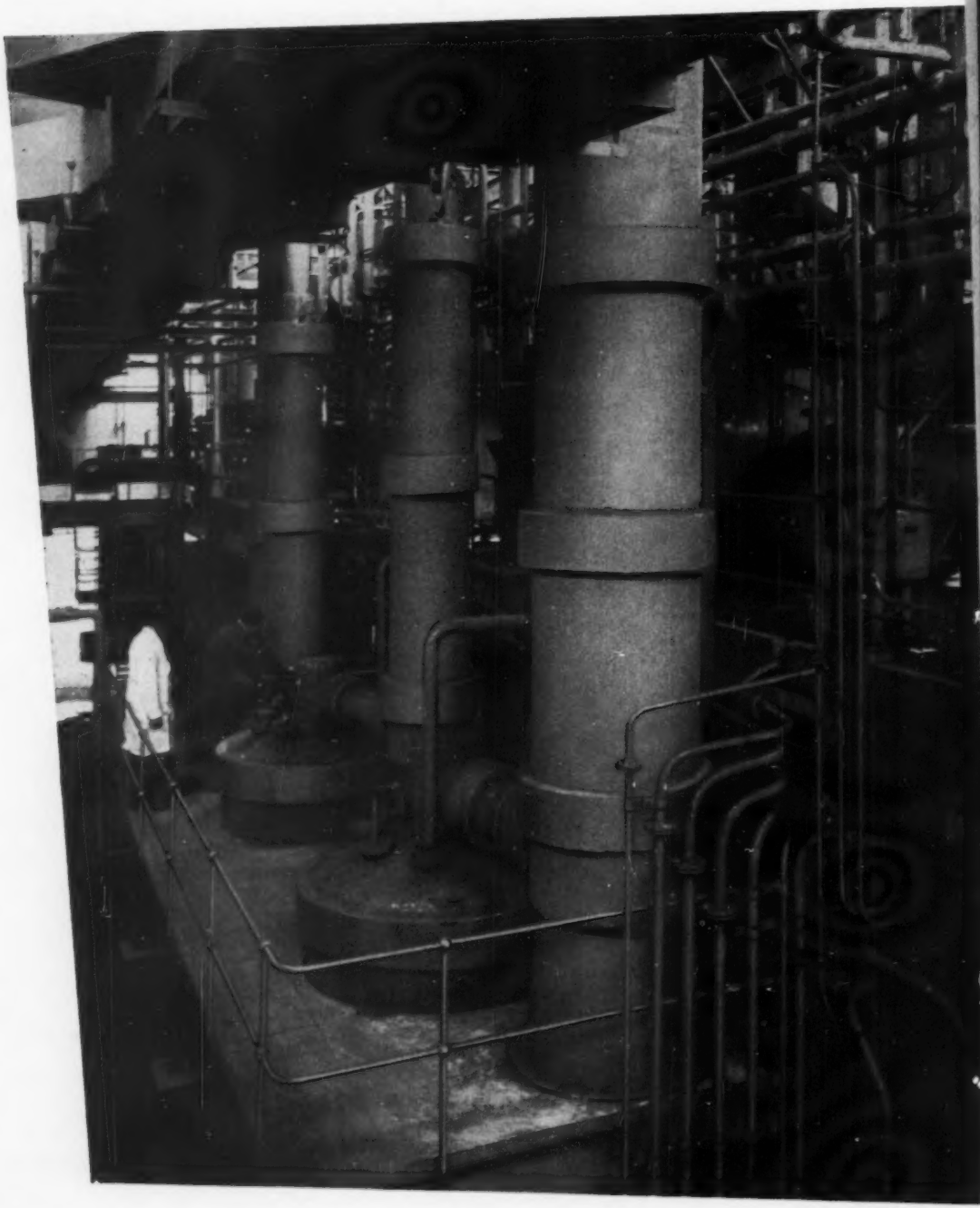

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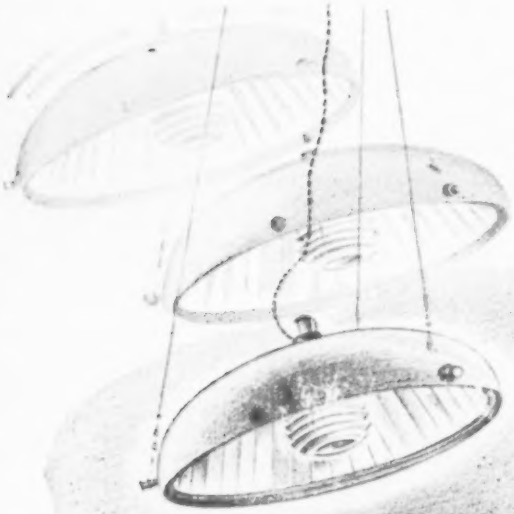

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BRITISH JOURNAL OF ANAESTHESIA

VOL. XXXII, No. 7 JULY 1960

EDITORIAL

HALOTHANE AND THE CLOSED CIRCUIT

SINCE halothane was introduced, the anaesthetic world has been roughly divided into those who hold this new agent as a panacea for all the anaesthetists' troubles—the perfect anaesthetic—and those who regard it with the greatest suspicion as differing little from other potent anaesthetics in that it is a drug having widespread effects and affecting every system in the body. In between these two extremes, there have been those who have quietly explored its possibilities, particularly as a supplement to, or substitute for, nitrous oxide anaesthesia in those operations where spontaneous respiration is permissible. It is largely for this last indication that it seems likely to find its place. The matter of expense, however, remains and it is universally agreed that, if possible, this is an agent which should be used with rebreathing and carbon dioxide absorption. Apart from expense, if used in such a way that there is an appreciable leakage into the surrounding atmosphere, it has been known to have effects on those working in the Operating Theatre. Headaches, for example, have occurred among the anaesthetic personnel when this agent has been used.

Whatever halothane has done, it has drawn attention to many of the problems which have been present ever since the introduction of the gaseous and volatile anaesthetic agents—problems for example concerning the uptake and elimination of these agents and now particularly problems concerning the possibility of a build-up when administered through vaporizers in the closed circuit. These are, of course, questions which apply to all agents, and the principles enunciated for halothane will equally be applicable to other circumstances. As Dr. Galloon has written in the summary to his paper "Although the effects of the above changes have been described throughout in terms of halothane concentration, the general principles outlined and the conclusions drawn apply to the vaporization of any liquid anaesthetic with a high enough rate of uptake in these types of anaesthetic systems." Professor Mushin and his colleagues at Cardiff have done a great service in exploring this question thoroughly and the results of their theoretical and experimental work are presented in the first three papers of this issue. It is good to see one more advance in our understanding of the procedures in which we are engaged daily.

Brit. J. Anaesth. (1960) **32**, 298

THE CONCENTRATION OF ANAESTHETICS IN CLOSED CIRCUITS, WITH SPECIAL REFERENCE TO HALOTHANE

I : THEORETICAL STUDY

BY

W. W. MAPLESON

Department of Anaesthetics, Welsh National School of Medicine, Cardiff

IN a non-rebreathing circuit, that is one in which all the patient's expirations are voided to the atmosphere, the concentration of anaesthetic which the patient inspires is usually known, at least roughly, from the setting of the controls on the anaesthetic machine. In a closed circuit, on the other hand, many other factors affect the inspired concentration, which is therefore not so readily discovered. In this paper the effects of these factors are studied theoretically, while many of the findings are confirmed experimentally by Galloon (1960) in the second paper of this series. The conclusions reached in these two papers have made possible the discussion of their clinical implications by Mushin and Galloon (1960) in the final paper.

Because of the current interest in halothane the detailed results are given mainly in terms of that agent, but in this paper an entirely general solution is derived which can be applied to any anaesthetic for which sufficient data are available.

THEORY

General

The basic principle of the theory is that, if the anaesthetic system is in a steady state, then the volume of any gas or vapour which enters the circuit in a given period of time must be equalled by the volume which leaves during the same period—either as a flow out to the atmosphere, or by uptake into the patient's body, or by absorption (as of carbon dioxide by soda lime).

A completely steady state will be reached only when the body is saturated with anaesthetic at the inspired tension, and when, therefore, the uptake of anaesthetic has ceased. However, the experiments of Haggard (1924) with ether, Severinghaus (1954) with nitrous oxide, and Duncan and Raventós (1959) with halothane (together with some measurements made with halothane in this Department) show that after a steep initial drop the rate of uptake falls so slowly that it does not reach zero for several hours or even days. Furthermore Kety's (1951) theory of inert-gas exchange suggests that the same will be true of all inhalation anaesthetics. Therefore it would be of little practical value to calculate the inspired concentrations that would exist with no uptake of anaesthetic. However, the very slowness with which the rate of uptake falls means that, at any given moment, except perhaps for the first few minutes of administration, the circuit is in a near enough steady state (with a nearly steady rate of uptake for the basic principles of the theory to be applied.

This is done in the appendix where equations are derived (Nos. 12 to 15) which express the inspired concentration, or the alveolar concentration, in terms of various parameters. These equations have been solved for a large number of specific cases and typical results are presented in graphical form in the next section. These graphs show the way in which the inspired (or sometimes the alveolar) concentration varies with the different parameters.

One of these parameters is the rate of uptake. As this varies with time the inspired concentration also varies with time. This is not shown graphically but the general nature of the variation can be deduced qualitatively assuming that the vaporizer setting and the fresh-gas flow are kept constant throughout the administration. The experimental work of Haggard, Severinghaus, and Duncan and Raventós, together with the measurements made in

this Department, shows that the rate of uptake is relatively high initially and then falls—fairly rapidly at first and then more and more slowly. From figure 9, which shows that the alveolar concentration rises as the rate of uptake falls, it can be deduced that the above pattern of uptake will lead to inspired concentrations which are initially low and then rise—fairly rapidly at first and then more and more slowly. In drawing the graphs (other than figure 9) a single rate of uptake has been used throughout and the value chosen is that which is to be expected at about 40 to 50 minutes after induction. The nature of the variation of rate of uptake with time is such that for any given set of circumstances the inspired concentration will approach the value indicated by the graphs within about 20 minutes, become equal to it after about 40 or 50 minutes, and yet not greatly exceed it even after 2 hours.

Assumptions

Some of the assumptions made in working out the theory may not always be justified. These are discussed below.

It is assumed that there are no leaks in the circuit. An outward leak would merely tend to act as an expiratory valve and so be unimportant, but an inward leak would be roughly equivalent to an increase in the fresh-gas flow.

It is assumed that when any gas escapes through the expiratory valve it has the composition of mixed-expired gas. This can only be true if the volume which escapes at each expiration is no greater than the tidal volume. This limits the theory to conditions where the input flow of fresh gas is less than the patient's minute-volume ventilation. Even so there are two situations which may lead to different results. If the expiratory valve is close to the patient, the escape, which occurs towards the end of the expiratory phase, may consist more nearly of alveolar gas; if this is so, less anaesthetic is lost and the inspired concentration becomes higher than that calculated. Again, if the valve is close to the fresh-gas inlet some fresh gas may escape directly through the valve. In this case the inspired concentration may be higher or lower than that calculated, depending on the circumstances, but it will be that concentration which corresponds to the net inflow of fresh gas, which is necessarily less than the flow rate shown by the flowmeters. In both cases the errors introduced can, in certain circumstances, be substantial—but the general trend of the results will not be altered.

It is assumed that only one anaesthetic is administered, together with oxygen. If two anaesthetics are administered simultaneously an exact determination of the inspired concentration of either would require a new equation. However, if halothane is combined with nitrous oxide the uptake of the latter is so small after the first few minutes of administration that, in calculating the inspired concentration of halothane, the flow of nitrous oxide may be regarded merely as an addition to the input flow of oxygen. The graphs in the results section are therefore valid whether the fresh-gas flow is pure oxygen or a mixture of oxygen and nitrous oxide.

It is assumed that the setting of the vaporizer and the fresh-gas flow are kept constant throughout the administration. However, if the setting of the vaporizer is increased after a time, then the inspired concentration will rise, probably fairly rapidly, to almost the same level that it would have reached if the vaporizer had been at the higher setting from the start. On the other hand, if the vaporizer is turned down, or the fresh-gas flow adjusted to produce a drop in inspired concentration, the degree of saturation of the body with anaesthetic will then be higher than if the new settings of the controls had existed throughout the administration. Therefore the rate of uptake will be lower than assumed in this theory and the resultant inspired concentration higher than predicted in the following graphs. The magnitude of the discrepancy will depend on the circumstances: if the controls are set to produce a high inspired concentration for an hour or more and then turned down, the subsequent inspired concentration may be appreciably above that predicted by the theory; on the other hand, the use of a high inspired concentration for just a few minutes to speed induction will not have much effect on the subsequent concentration.

Circuits

From the point of view of this theory closed circuits may be divided into two classes.

(1) *The vaporizer outside the breathing circuit* (*V.O.C.*). The circuit may be either a to-and-fro or a circle system; the essential feature is that the

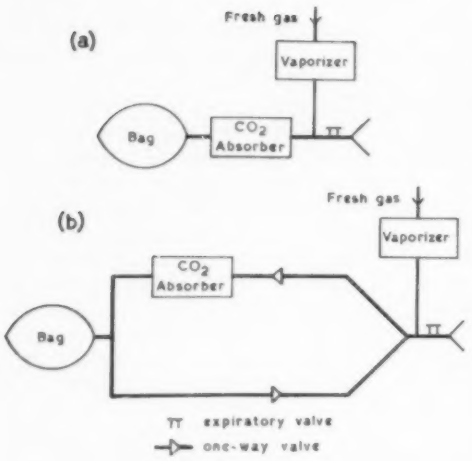

FIG. 1

Diagrammatic representation of two examples of closed circuits in which the vaporizer is *outside* the breathing circuit (V.O.C.): (*a*) a to-and-fro system; (*b*) a circle system (the fresh gas may enter the breathing circuit at other points than the one shown).

vaporizer is placed in the fresh-gas supply line so that the circuit receives a steady supply of anaesthetic (fig. 1).

(2) *The vaporizer inside the breathing circuit (V.I.C.).* The circuit is invariably a circle system; but the essential feature is that the vaporizer is somewhere in the breathing circuit so that the patient's inspirations or expirations pass through it. The performance also depends partly on whether or not any of the fresh gas passes through the vaporizer before reaching the patient; this, in turn, depends on the relative positions of the components of the system. The arrangements in three common systems of the class are shown in figure 2 and their performance is considered in the appendix.

Parameters involved

The following parameters affect the inspired and the alveolar concentrations:

(1) *The "vaporizer concentration".* This is a convenient abbreviation for the quantity defined in the appendix as "the concentration delivered by the vaporizer when fresh gas, containing no vapour, passes through it". It is also, of course, the concentration delivered to a closed circuit by a vaporizer outside the circuit—since then only fresh gas does pass through the vaporizer. It is not, however, the concentration emerging from a vaporizer situated inside a breathing circuit since the gas which enters a vaporizer in that position already contains some anaesthetic from the patient's expirations. The significance of the term in this case will become apparent in the results section; meanwhile, it may be noted that in most circumstances the vaporizer concentration is approximately equal to the concentration added to the gas in passing through a vaporizer inside the breathing circuit (see appendix).

This parameter, the vaporizer concentration, is nominally under the control of the anaesthetist by means of a tap, but it is important to remember that in most vaporizers the concentration varies with temperature and with the flow through the vaporizer as well as with the tap setting.

(2) *The input flow of fresh gas.* This is under the control of the anaesthetist. Values above 3 l./min

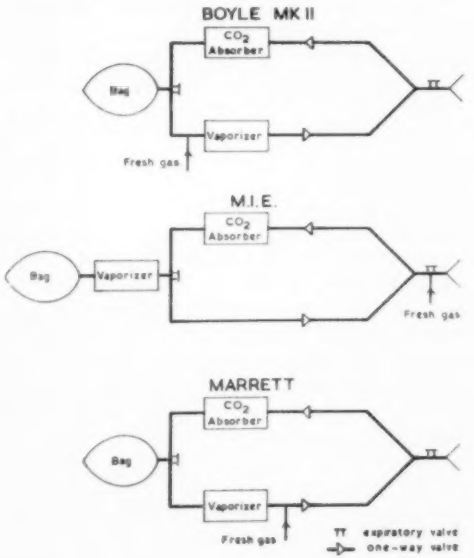

FIG. 2

Diagrammatic representation of three examples of circle systems with the vaporizer *inside* the breathing circuit (V.I.C.).

are not included in the results because at high fresh-gas flows the assumption that it is mixed-expired gas which escapes through the expiratory valve is no longer valid.

(3) *The patient's total minute-volume ventilation.* A value of 5 l./min has been used for many of the graphs but the effect of variation over the range from 2 to 10 l./min is also included.

(4) *The alveolar ventilation.* This is related to the total ventilation by the respiratory rate and the deadspace. The values used in plotting the graphs are 20 respirations/min for the rate and 130 ml for the deadspace (but see appendix). The use of other values would produce somewhat different inspired concentrations but would not alter the general pattern of results.

(5) *The average flow rate through the vaporizer.* If the vaporizer is outside the breathing circuit this is simply the fresh-gas flow rate. If the vaporizer is inside the breathing circuit it is primarily the patient's ventilation which flows through it, but the fresh-gas flow may also have some effect (see appendix).

(6) *The patient's oxygen consumption.* This is assumed to be 200 ml/min but variation from 100 to 300 ml/min would have very little effect on the results.

(7) *The rate of uptake of anaesthetic by the patient.* The values used in constructing the graphs and in drawing general conclusions are shown in table I. The derivation of the values is explained in the appendix.

TABLE I

Approximate rates of uptake of anaesthetics in the period from 20 minutes to 2 hours after induction.

Anaesthetic	Rate of uptake in ml of gas or pure vapour per minute for each per cent alveolar concentration
Ethylene	0.5
Cyclopropane	1.5
Nitrous oxide	2
Halothane	15
Chloroform	50
Ether	100

RESULTS

The graphs of figures 3 to 8 show the results of solving equations (13) and (14) in the appendix for the case of halothane. Each graph shows the way in which the inspired or alveolar concentrations of halothane vary with some parameter of closed-circuit anaesthesia when the other parameters are kept constant. Sometimes the performance of a non-rebreathing circuit is included for comparison. As already explained, the concentrations shown are approached within 20 minutes of induction but not greatly exceeded for at least 2 hours. The graphs apply only to halothane, but some indication is given in the text of how the circuits behave with other anaesthetics.

Experimental confirmation of many of the results will be found in the second paper of this series (Galloon, 1960) and a discussion of their clinical implications in the final one (Mushin and Galloon, 1960).

Vaporizer concentration

Figure 3 compares the two main types of closed circuit and a non-rebreathing circuit in terms of the variation of inspired concentration with

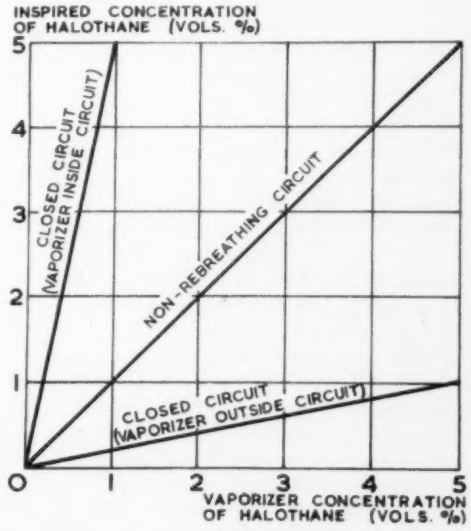

FIG. 3

Variation of inspired concentration of halothane with vaporizer concentration (concentration leaving the vaporizer when fresh gas passes through it) for three circuits: a non-rebreathing circuit, a closed circuit with the vaporizer outside the breathing circuit, and a closed circuit with the vaporizer inside the breathing circuit. For the closed circuits: fresh-gas flow = 0.2 l./min (basal oxygen only); patient's total minute-volume ventilation = 5 l./min.

vaporizer concentration. With a non-rebreathing circuit it is true for all anaesthetics that the inspired concentration is equal to the concentration coming from the vaporizer. This is shown on the graph by the straight line at a slope of 45°. In the closed circuits the inspired concentration may be higher or lower than the vaporizer concentration, depending on circumstances, but one is still proportional to the other in the range shown.

When the vaporizer is inside the breathing circuit and the fresh-gas flow is small, the inspired concentration is always greater than the vaporizer concentration. This is because the gas which enters the vaporizer already contains some anaesthetic from the patient's expirations and therefore the concentration which leaves it, to be inspired by the patient, is higher than the vaporizer concentration—the concentration which would leave it if only fresh gas entered it. The uppermost line in figure 3 shows that, in the case of halothane, and when the supply of fresh gas is confined to a basal flow of oxygen, the inspired concentration is as much as five times the vaporizer concentration. For an anaesthetic with a higher rate of uptake than halothane, the discrepancy is less: for example, with ether, the inspired concentration may be no more than twice the vaporizer concentration.

The practical significance of this difference between inspired and vaporizer concentration is as follows. A vaporizer which in a laboratory calibration showed a maximum vaporizer concentration of 1.5 per cent halothane, as in the prototype of the Goldman (1959), or about 2 per cent halothane, as in one model of the Rowbotham (Newman, 1958), might be considered as inherently safe in a non-rebreathing circuit—where the inspired concentration is equal to the vaporizer concentration. But if such vaporizers were used inside a closed circuit (V.I.C.) full on, and with only a basal fresh-gas flow, the inspired concentrations could rise to 7.5 or 10 per cent halothane respectively.

When the vaporizer is outside the breathing circuit and the fresh-gas flow is small, the inspired concentration may be either greater or less than the vaporizer concentration (which is here the concentration in the fresh-gas supply line) depending on the rate of uptake of the anaesthetic. Roughly speaking, if the anaesthetic is not taken up very readily by the body—as is the case with nitrous oxide, cyclopropane and ethylene—it will tend to accumulate in the circuit while the oxygen is freely absorbed by the patient. Therefore the concentration inspired from the circuit is higher than that entering it from the fresh-gas supply line. This is a common feature of clinical experience with nitrous oxide and is usually explained by regarding the uptake as negligible. During the first few minutes of nitrous oxide anaesthesia, however, the uptake is high enough for the reverse to be true (Severinghaus, 1954): the anaesthetic is then taken up so readily that it is the oxygen which, relatively, is left to accumulate in the circuit; therefore the inspired concentration of anaesthetic is less than that entering the circuit by the supply line. This is also the case with halothane and ether—and probably all volatile anaesthetics in current use—except that with these the rate of uptake stays high enough to keep the inspired concentration below the supply concentration throughout many hours' administration. The lowest line in figure 3 shows that, in the case of halothane, when the supply of fresh gas is confined to a basal flow of oxygen, the inspired concentration is only about one-fifth of the vaporizer concentration. With ether under the same conditions it might be as little as one-tenth. This paragraph and the preceding one explain Newman's (1958) statement for halothane that a vaporizer which is to be used outside the breathing circuit must have a high "efficiency" (of vaporization) whereas one that is used inside the breathing circuit need have only a low efficiency.

Fresh-gas flow

The way in which the inspired concentration of halothane varies with fresh-gas flow is illustrated in detail in figures 4 and 5. Figure 4 is for the case of the vaporizer outside the breathing circuit and shows a number of curves, each for a different input, or vaporizer, concentration. As was seen in figure 3, when there is only a basal fresh-gas flow the inspired concentration is much less than the corresponding vaporizer concentration. As the fresh-gas flow is increased, however, the inspired concentration gradually increases—at first rapidly, then more and more slowly—until at a fresh-gas flow of 2 to 3 l./min it is not much less than the vaporizer concentration. For most other anaesthetics, the same general pattern of results would be found and the curves would be at about the same height at the higher fresh-gas flows; but as the

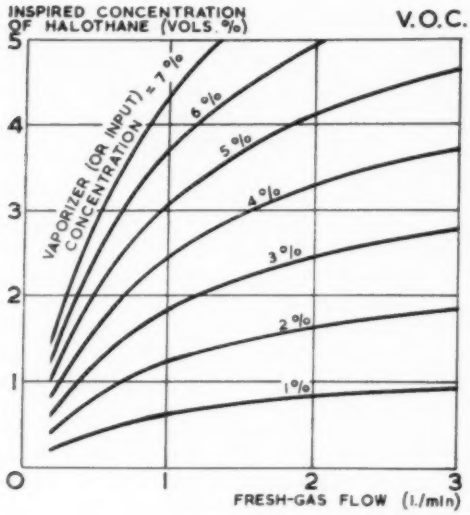

FIG. 4

Variation of inspired concentration of halothane with fresh-gas flow for various vaporizer concentrations. Vaporizer *outside* the breathing circuit. Patient's total ventilation = 5 l./min.

fresh-gas flow was reduced the curves would fall more steeply for those anaesthetics with a high rate of uptake, e.g. ether, or less steeply for those with a low rate of uptake. If the rate of uptake is very low, as with nitrous oxide (after the first few minutes of administration), cyclopropane, or ethylene, the slope of the lines is reversed: the inspired concentration is higher than the input concentration at low fresh-gas flows and decreases as the flow is increased.

Figure 5 is for the case of the vaporizer inside the breathing circuit and shows the reverse of the pattern of figure 4. At a basal fresh-gas flow the inspired concentration of halothane is much greater than the vaporizer concentration. As the fresh-gas flow is increased the inspired concentration falls—at first rapidly, then more and more slowly—until, at 3 l./min, the inspired concentration is only about $1\frac{1}{2}$ times the vaporizer concentration. The curves have been calculated on the assumption that all the fresh gas passes through the vaporizer before reaching the patient, as in the Boyle Mark II circle system.

If the fresh gas tends to go directly to the patient, as in the M.I.E. and Marrett systems, this may reduce the inspired concentrations at a flow of 3 l./min by up to a third; but the reduction becomes negligible at smaller flows. In the extreme case of a very low ventilation (say 3 l./min) combined with a high fresh-gas flow (also 3 l./min) the circuit may behave very nearly as a non-rebreathing system so that the patient receives nearly pure fresh gas direct from the supply line. The inspired concentration would then be almost zero whatever the vaporizer setting.

The same general pattern of results will be found for all anaesthetics. The only difference is that for those with a higher rate of uptake than halothane (ether, chloroform), the inspired concentrations will be generally lower—closer to the vaporizer concentrations and changing less rapidly with fresh-gas flow—while for those with a lower rate of uptake the reverse will be true.

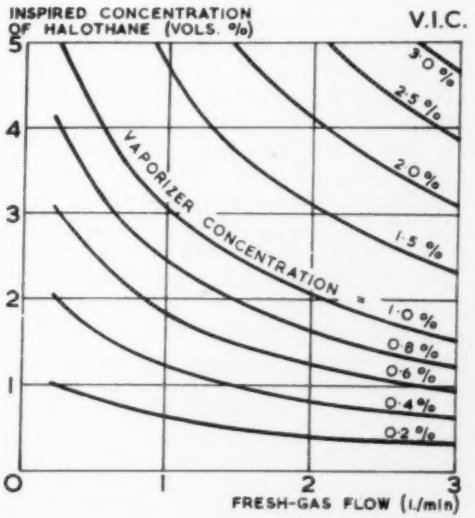

FIG. 5

Variation of inspired concentration of halothane with fresh-gas flow for various vaporizer concentrations. Vaporizer *inside* the breathing circuit and all fresh gas passing through the vaporizer before reaching the patient. Patient's total ventilation = 5 l./min.

Ventilation

In all the graphs so far described (figures 3 to 5) it has been assumed that the patient's total minute-volume ventilation was 5 l./min. In the next three graphs (figures 6 to 8) the effects of changes in the ventilation are illustrated.

Figure 6 refers to the case where the vaporizer is outside the breathing circuit. The inspired concentration of halothane has been plotted against the total (and corresponding alveolar) ventilation for a number of different input flows of fresh-gas. For each flow an input concentration has been chosen such that when the total ventilation is 5 l./min the inspired concentration is 1.5 per cent. For example: for a fresh-gas flow of 0.2 l./min a vaporizer concentration of 7.3 per cent is needed; while for a flow of 2 l./min a concentration of only 1.8 per cent is needed. Thus an inspired concentration of 1.5 per cent with a total ventilation of 5 l./min is regarded, for the sake of argument, as a sort of norm which might be established early in anaesthesia. If the ventilation were then to change, the inspired concentration would change according to the line on the graph which corresponded to the particular input flow in use. If, for instance, the input flow is so large that there is no rebreathing then the inspired concentration is not altered by changes in the ventilation as is shown by the horizontal line on the graph (E, fig. 6). On the other hand, if only a basal fresh-gas flow (0.2 l./min) is used (A, fig. 6), any decrease in ventilation produces a very steep increase in inspired concentration, while an increase in ventilation produces a less marked decrease.

However, this graph needs to be interpreted with caution. Unlike the parameters that have been considered previously, changes in ventilation produce changes in the relationship between the inspired and alveolar concentrations. Since depth of anaesthesia is more closely related to the alveolar than to the inspired concentration, it is more useful to look at the variations in alveolar concentration. These are shown, for the case of the vaporizer outside the breathing circuit, in figure 7. Here, for each input flow, the vaporizer concentration has been chosen so that when the total ventilation is 5 l./min. the alveolar concentration is 1 per cent. (This corresponds very nearly to an inspired concentration of 1.5 per cent at a ventilation of 5 l./min.)

It will be seen that the condition of no rebreathing (curve E), in which the inspired concentration was independent of ventilation, is accompanied by an alveolar concentration which falls off as the ventilation decreases. This is because the reduced ventilation is less efficient in conveying the halothane from the circuit to the alveoli.

At the other extreme: the condition of only basal fresh-gas flow, which led to a steep increase in inspired concentration as the ventilation fell, gives an alveolar concentration which is unaffected by ventilation. This arises from the fact that with a basal fresh-gas flow the circuit is completely closed and therefore, in the steady state, all the halothane that enters *must* be absorbed at the alveoli. Therefore, when the ventilation falls, the circuit (or inspired) concentration builds up so that the gradient of concentration from the circuit to the alveoli is enough to compensate for the reduced ventilation and to maintain the original alveolar concentration and rate of uptake.

For other anaesthetics different input and inspired concentrations would generally be required for the same alveolar concentrations, but when the fresh-gas flow was basal the alveolar concentration would always be independent of ventilation. However, at high fresh-gas flows, or in non-rebreathing circuits, a high rate of uptake would prevent the alveolar concentration approaching the inspired concentration until the ventilation was very large; therefore alveolar concentration would increase with ventilation over the whole of the practical range instead of flattening out above about 5 l./min as the curves for halothane do. Conversely, a low rate of uptake (when the fresh-gas flow is high) would allow the alveolar concentration to approach the inspired concentration even at quite low ventilations; therefore the alveolar concentrations would then be independent of the ventilation except for falling off at very low ventilations.

Figure 8 shows what happens when the vaporizer is inside the circuit and all the fresh gas passes through the vaporizer.* Again the alveolar concentration is plotted against the ventilation for

* If the fresh gas goes directly to the patient the pattern will be much the same for fresh-gas flows up to 1 or 2 l./min. Higher fresh-gas flows will tend to eliminate rebreathing at the lower ventilations and therefore make the alveolar concentration approach zero (see above).

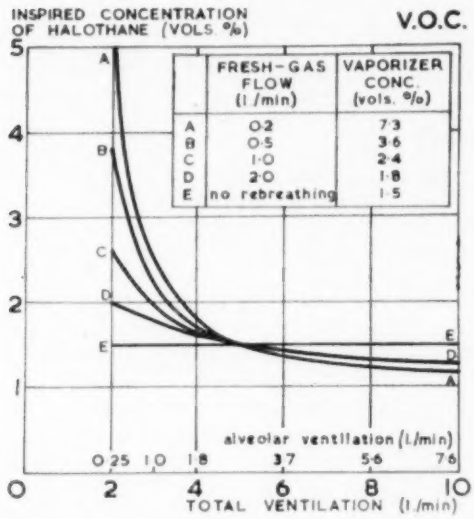

FIG. 6

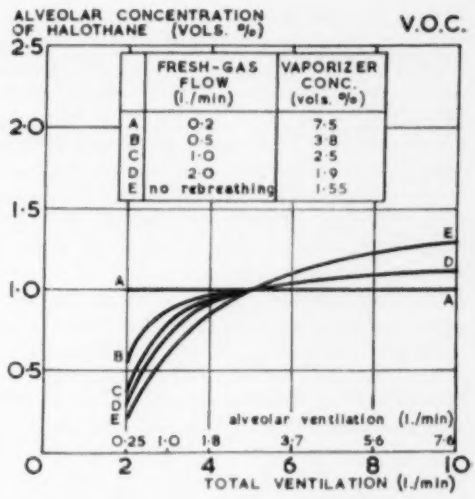

FIG. 7

FIG. 6

Variation of inspired concentration of halothane with total and alveolar ventilation for various fresh-gas flows. Vaporizer *outside* the breathing circuit. For each fresh-gas flow the input, or vaporizer, concentration has been chosen so that when the total ventilation is 5 l./min the inspired concentration is 1.5 per cent.

FIG. 7

Variation of alveolar concentration of halothane with total and alveolar ventilation for various fresh-gas flows. Vaporizer *outside* the breathing circuit. For each input flow the input, or vaporizer, concentration has been chosen so that when the total ventilation is 5 l./min the alveolar concentration is 1 per cent (corresponding very nearly to an inspired concentration of 1.5 per cent). (Note change of concentration scale from previous graphs.)

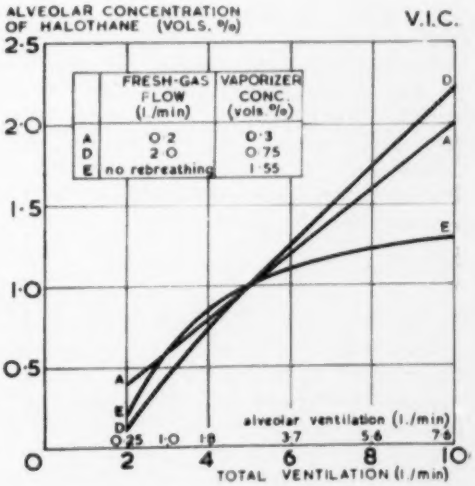

FIG. 8

FIG. 8

Variation of alveolar concentration of halothane with total and alveolar ventilation for various fresh-gas flows. Vaporizer *inside* the breathing circuit and all fresh gas passing through the vaporizer before reaching the patient. For each fresh-gas flow the vaporizer concentration has been chosen so that when the total ventilation is 5 l./min the alveolar concentration is 1 per cent.

various fresh-gas flows, and again for each flow a vaporizer concentration has been chosen such that when the total ventilation is 5 l./min the alveolar concentration is 1 per cent. The condition of no rebreathing gives the same result as before, of course, but at the lower fresh-gas flows the alveolar concentration changes much more rapidly with ventilation than when the vaporizer is outside the breathing circuit. In fact, with a basal fresh-gas flow, the alveolar concentration is proportional to the total ventilation (A, fig. 8). (This is because, with a basal fresh-gas flow the circuit is completely closed and therefore the rate of uptake must equal the rate of vaporization; the rate of uptake is proportional to the alveolar concentration and, with the vaporizer inside the circuit, the rate of vaporization is proportional to the ventilation; therefore the alveolar concentration is proportional to the ventilation.) The performance is very little different even for a fresh-gas flow as high as 2 l./min (D, fig. 8).

For anaesthetics with lower rates of uptake than halothane the alveolar concentration still increases in proportion to the total ventilation when the fresh-gas flow is basal. But when the fresh-gas flow is high, or when a non-rebreathing circuit is used, the alveolar concentration is practically independent of ventilation: it falls off only when the alveolar ventilation drops very nearly to zero. For anaesthetics with high rates of uptake, on the other hand, the steep increase of alveolar concentration with ventilation persists for all fresh-gas flows and even in non-rebreathing circuits (over the range of ventilation shown).

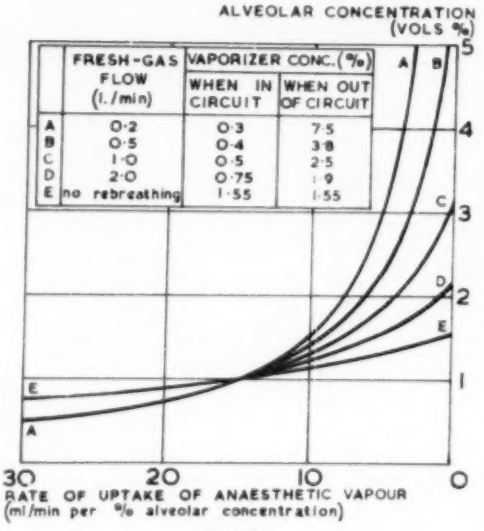

FIG. 9

Variation of alveolar concentration with rate of uptake of anaesthetic for various fresh-gas flows. Vaporizer inside *or* outside the breathing circuit. (Note: this graph shows the way in which the alveolar concentration depends on the rate of uptake when other conditions are kept constant. It does not show how the uptake depends on the alveolar concentration—this problem is discussed in the appendix.) As the rate of uptake of anaesthetic decreases (as it does, with time, in a given patient) so the alveolar concentration rises. For each fresh-gas flow the vaporizer concentration has been chosen so that the alveolar concentration is 1 per cent when the rate of uptake is 15 ml/min for each per cent alveolar concentration. Patient's total ventilation = 5 l./min.

Rate of uptake of anaesthetic

In all the graphs so far described (figures 3 to 8) the rate of uptake of halothane vapour has been assumed to be 15 ml/min for each per cent alveolar concentration. But the uptake does, of course, vary with time, and from patient to patient, so figure 9 is included to show the way in which the alveolar concentration depends on the rate of uptake for various fresh-gas flows. (The way in which the uptake depends on the alveolar concentration is a different matter and is discussed in the appendix.) The curves apply to both classes of closed circuit (vaporizer inside or outside the breathing circuit) provided the vaporizer concentrations are kept constant at the values shown. These concentrations have been chosen so that at each fresh-gas flow the alveolar concentration is 1 per cent when the rate of uptake is 15 ml/min for each per cent alveolar concentration (assuming a total ventilation of 5 l./min). It is seen that a decrease in the uptake constant produces an increase in the alveolar concentration; the increase is very marked with a basal fresh-gas flow (curve A) but very slight with a high fresh-gas flow. The corresponding changes in inspired concentration are similar but in the non-rebreathing condition the inspired concentration is independent of the uptake constant.

Since the rate of uptake gradually decreases during anaesthesia (as the patient becomes more and more saturated with anaesthetic) there will necessarily be a gradual increase in alveolar con-

centration if all other conditions remain the same. With a non-rebreathing circuit the effect would be very small, but with a low fresh-gas flow it might be big enough to be detected clinically in a long case.

In addition, there is reason to expect that in children the rate of uptake falls off more rapidly than in adults: therefore it might be expected that the alveolar concentration, and hence the depth of anaesthesia, would increase more quickly in children than in adults.

The graphs of figures 6 to 9 would still show the same pattern if they were replotted for other vaporizer concentrations. In fact, over the range of concentrations which are of interest with halothane, the existing graphs would still be true if the scales of inspired or alveolar concentration were simply multiplied by some factor, provided the vaporizer concentrations mentioned were multiplied by the same factor.

SUMMARY

A theoretical study has been made of the concentrations of anaesthetic gases and vapours in closed circuits. Two classes of closed circuit are distinguished: those with the vaporizer inside the breathing circuit (V.I.C.) and those with it outside (V.O.C.). The way in which the inspired and alveolar concentrations vary with different parameters in these two circuits and in a non-rebreathing circuit is shown by equations (in the appendix) and by graphs.

ACKNOWLEDGMENTS

In making theoretical calculations of this sort it is easy to become involved in obscurities which are mathematically fascinating but of little or no clinical interest. I am extremely grateful therefore to Professor William W. Mushin for his constant advice in guiding the work in clinically useful directions. I am also very grateful to Dr. S. Galloon for many helpful discussions; the regular comparison of his experimental results with my theoretical findings proved a constant inspiration in developing the work. I am grateful also to Messrs. Imperial Chemical Industries Ltd., for answering many questions about the physical properties of halothane. Finally my thanks are due to Dr. P. W. Thompson and Dr. D. Waters for reading the manuscript and making helpful comments.

APPENDIX

Before proceeding to the main part of the theory it is necessary to establish a relationship between the rate of uptake of anaesthetic by the patient at the alveoli, $\dot{V}_{p,an}$*, and the fractional alveolar concentration, F_A. In doing this, much use has been made of Kety's (1951) review of theories of inert-gas exchange.

The rate of uptake of anaesthetic is the rate at which it is removed from the alveoli by the blood, which is $\dot{Q}(C_a - C_{\bar{v}})$ (assuming no shunts) where $\dot{Q}$ is the cardiac output, C_a the arterial concentration, and $C_{\bar{v}}$ the mixed-venous concentration. That is:

$$\dot{V}_{p,an} = \dot{Q}(C_a - C_{\bar{v}}),$$
$$= C_a\dot{Q}(1 - C_{\bar{v}}/C_a). \quad (1)$$

Assuming that the arterial concentration is in equilibrium with the alveolar concentration (Kety, 1951),

$$C_a = \lambda F_A \quad (2)$$

where λ is the blood/gas partition coefficient for the anaesthetic vapour at body temperature. Substituting (2) in (1):

$$\dot{V}_{p,an} = \lambda\dot{Q}(1 - C_{\bar{v}}/C_a)F_A. \quad (3)$$

For any given anaesthetic λ is a constant. $C_{\bar{v}}/C_a$ is a measure of the saturation of the venous return and will obviously increase with time; but there seems no reason to expect it to depend on the alveolar concentration. With some anaesthetics $\dot{Q}$ may decrease as F_A (and hence C_a) increases but not enough is known about this to take account of it mathematically. It is preferable to write

$$\dot{V}_{p,an} = kF_A, \quad (4)$$

where k is a constant of proportionality which depends on the degree of saturation of the venous return, and therefore decreases with time, and which, with some anaesthetics, may decrease at higher values of F_A because of a resultant fall in cardiac output.

If the circuit is in a steady state the rate at which anaesthetic vapour enters must equal the rate at which it leaves. This can be applied in three ways. Firstly, in the circuit as a whole:

$$\dot{V}_{in,an} = \dot{V}_{out,an} + \dot{V}_{p,an}, \quad (5)$$

where $\dot{V}_{in,an}$ = rate of input (or vaporization) of anaesthetic and $\dot{V}_{out,an}$ = rate of output of anaesthetic vapour through the expiratory valve. Secondly, in the lungs:

$$F_I\dot{V} = F_{\bar{E}}\dot{V} + \dot{V}_{p,an}, \quad (6)$$

where $\dot{V}$ = patient's total minute-volume ventilation, F_I = inspired concentration of anaesthetic, $F_{\bar{E}}$ = mixed-expired concentration. Finally, in the alveoli:

$$F_I\dot{V}_A = F_A\dot{V}_A + \dot{V}_{p,an}, \quad (7)$$

where $\dot{V}_A$ = alveolar ventilation. Eliminating F_A from (4) and (7) and solving for $\dot{V}_{p,an}$:

$$\dot{V}_{p,an} = \theta F_I, \quad (8)$$

$$\text{where } \theta = k\dot{V}_a/(k + \dot{V}_a). \quad (9)$$

If the outflow through the expiratory valve is assumed to consist of mixed-expired gas (see main part of paper) then its fractional concentration, $F_{\bar{E}}$, can be equated to the anaesthetic outflow, $\dot{V}_{out,an}$, divided by the total outflow, $\dot{V}_{out,t}$:

$$F_{\bar{E}} = \frac{\dot{V}_{out,an}}{\dot{V}_{out,t}}$$

* The notation used here is basically that of the Pappenheimer (1950) system (Campbell, 1957), modified to avoid conflict with British Standard 1991, Part I (1954), and slightly simplified when this achieves brevity without introducing ambiguity.

Water vapour and carbon dioxide will form only a small part of the total outflow (assuming efficient absorption of carbon dioxide) so the total outflow will be almost the same as the oxygen outflow, $\dot{V}_{out,O}$, plus the anaesthetic outflow, $\dot{V}_{out,an}$:

$$F_E = \frac{\dot{V}_{out,an}}{\dot{V}_{out,O} + \dot{V}_{out,an}}. \quad (10)$$

Substituting (8) and (10) in (6) and solving for $\dot{V}_{out,an}$:

$$\dot{V}_{out,an} = \frac{\dot{V}_{out,O}F_I(1 - \theta/\dot{V})}{1 - F_I(1 - \theta/\dot{V})}. \quad (11)$$

Substituting (8) and (11) in (5) and solving for F_I:

$$\theta(1 - \theta/\dot{V})F_I^2 - \{(\dot{V}_{out,O} + \dot{V}_{in,an})(1 - \theta/\dot{V}) + \theta\}F_I + \dot{V}_{in,an} = 0. \quad (12)$$

This is a quadratic which can be solved to give F_I in terms of various parameters. If F_I is small it is possible to obtain a simpler equation by neglecting the term involving F_I in the denominator of (11). This leads to:

$$F_I = \frac{\dot{V}_{in,an}}{\dot{V}_{out,O}(1 - \theta/\dot{V}) + \theta}. \quad (13)$$

The error involved in doing this, expressed as a percentage of the true value of F_I, is about equal to the percentage inspired concentration itself. For clinical accuracy, therefore, (13) may be used for inspired concentrations up to about 20 per cent.

For both (12) and (13) the corresponding values of alveolar concentration can be derived by combining (4), (8) and (9):

$$F_A = F_I\dot{V}_A/(k + \dot{V}_A). \quad (14)$$

This equation is true for all circuits since its derivation involves only factors within the lungs.

Equations (12) to (14) represent a formal solution to the problem of determining the inspired and alveolar concentrations of anaesthetic in closed circuits under various conditions. To make use of the equations, however, it is necessary to express some of the parameters in terms of more readily determined factors.

$\dot{V}_{in,an}$, the rate of input of anaesthetic. For a gaseous anaesthetic $\dot{V}_{in,an}$ is simply the flow of fresh anaesthetic gas. For a volatile anaesthetic in the type of vaporizer in which liquid anaesthetic is added drop by drop $\dot{V}_{in,an}$ is the rate at which the liquid is added, expressed as a vapour flow rate instead of a liquid flow rate—provided, of course, that the liquid is vaporized as fast as it is added. With the type of vaporizer intended to contain a substantial quantity of liquid anaesthetic $\dot{V}_{in,an}$ is the rate of vaporization. If the vaporizer is outside the circuit

$$\dot{V}_{in,an} = \dot{V}_vF_v \quad (15)$$

where $\dot{V}_v$ is the total flow rate leaving the vaporizer and F_v is the "vaporizer concentration"—the concentration delivered by the vaporizer when fresh gas, containing no vapour, passes through it. If the vaporizer is inside the breathing circuit the rate of vaporization is modified by the fact that the gas entering the vaporizer already contains some anaesthetic from the patient's expirations. If it is assumed, as seems reasonable, that the increase in concentration which occurs as the gas passes through the vaporizer is a fixed fraction of the difference between the saturation concentration at the temperature of the liquid, F_S, and the concentration entering the vaporizer, F_{ent} then it can be shown that

$$\dot{V}_{in,an} = \dot{V}_vk' \frac{F_S - F_{ent}}{1 - F_{ent}}, \quad (16)$$

where k' is a constant and $\dot{V}_v$ is understood to be the average flow rate leaving the vaporizer. If only fresh gas were to pass through the vaporizer F_{ent} would equal zero and (16) would reduce to $\dot{V}_{in,an} = \dot{V}_vk'F_S$. But (15) would also then be true and therefore $k' = F_v/F_S$. Therefore (16) becomes

$$\dot{V}_{in,an} = \dot{V}_vF_v \frac{1 - F_{ent}/F_S}{1 - F_{ent}}. \quad (17)$$

If $F_{ent} \ll F_S$ this reduces to (15). Since F_{ent} will be equal to F_E (or even less if the patient's expirations are diluted by fresh gas before entering the vaporizer) it seems doubtful if the error introduced in equating (17) to (15) will be of clinical importance (say 20 per cent of the true value) for any of the volatile anaesthetics in current use. In practice, therefore, (15) may be taken to define $\dot{V}_{in,an}$ no matter whether the vaporizer is inside or outside the breathing circuit.

$\dot{V}_{out,O}$, the outflow of oxygen from the circuit. Since the circuit is assumed to be in a steady state $\dot{V}_{out,O}$ must equal the inflow of oxygen, $\dot{V}_{in,O}$, minus the patient uptake of oxygen, $\dot{V}_{p,O}$:

$$\dot{V}_{out,O} = \dot{V}_{in,O} - \dot{V}_{p,O} \quad (18)$$

$\dot{V}_A$, the alveolar ventilation. At the low tidal volumes that often occur under anaesthesia the relationship usually assumed between alveolar and total ventilation is invalid (Briscoe et al., 1954; Comroe et. al., 1955). Instead recourse has been made to the work of Gray, Grodins and Carter (1956). As some of their finer corrections are too small to be of consequence here, use is made of the simpler formula of Rohrer (1916) which they quote. In terms of ventilation the formula becomes:

$$\dot{V}_A = \dot{V}(1 - 0.5V_D/V_T)^2 \quad (19)$$

where V_D = "virtual deadspace" and V_T = tidal volume.

k, the rate of uptake of anaesthetic per unit fractional alveolar concentration. Experimental data on uptake are available for three dogs under ether anaesthesia (Haggard, 1924), for six patients under nitrous oxide (Severinghaus, 1954), and for nine patients under halothane (work done in this Department but not yet published), from which, by applying various of the foregoing equations, it is possible to estimate values of k for these anaesthetics. It has also been found possible to estimate k theoretically for any anaesthetic on the basis of its solubilities in water and in fat, and on the basis of existing knowledge of the processes of inert-gas exchange in the body. This has been done for six anaesthetics which might be used in a closed circuit. The details will be published in another paper but since the theoretically-derived values for ether, nitrous oxide and halothane agreed well with the experimental ones it seems reasonable to make use here of the theoretically derived values for the other anaesthetics. The values are not strictly applicable when a closed circuit is used, because the inspired concentration then gradually increases with time instead of remaining constant as was the case in the experimental work and as was assumed in the theoretical derivations. However, the discrepancy is likely to be small and in any case it is sufficient for the present work to choose convenient round figures which are reasonably representative of those to be expected in the period from 20 minutes to about 2 hours

after induction. These are given in the main part of the paper (table I).

$\dot{V}_v$, *the average flow rate through the vaporizer*. If the vaporizer is outside the breathing circuit the flow through it is simply the input flow of oxygen:

$$\dot{V}_v = \dot{V}_{in,O}. \tag{20}$$

If the vaporizer is inside the breathing circuit $\dot{V}_v$ is more closely related to the patient's ventilation. In the Boyle Mark II system (fig. 2) the average flow through the vaporizer is near enough equal to the total ventilation:

$$\dot{V}_v = \dot{V}. \tag{21}$$

In the M.I.E. and the Marrett systems the flow through the vaporizer is generally less than the total ventilation by some function of the input flow of oxygen. The function depends on the breathing pattern and on which expiratory valve is used, but in almost all circumstances $\dot{V}_v$ lies between $\dot{V}$ and $\dot{V} - \frac{1}{2}\dot{V}_{in,O}$:

$$\dot{V} \geqslant \dot{V}_v \geqslant \dot{V} - \tfrac{1}{2}\dot{V}_{in,O}. \tag{22}$$

NOTE ADDED IN PROOF

Since writing this paper Nunn (1960) has shown that under anaesthesia the relationship between total and alveolar ventilation is better described by an equation different from the one used here (which is valid for the conscious state). If the results shown in the graphs are recalculated according to Nunn's equation the changes produced are very small except in figures 6, 7 and 8. In these figures the values of alveolar ventilation shown are no longer valid but the curves relating inspired and alveolar concentrations to total ventilation are not materially affected for ventilations of 5 l./min upwards. As the ventilation falls below 5 l./min, however, the rises in inspired concentration and the falls in alveolar concentration are generally much less marked than shown except that the straight line A in figure 8 is unaffected.

Also since writing this paper, Messrs. M.I.E Ltd. have introduced a new circle system, the "Lincoln 800", in which the various components are arranged in the same sequence as shown for the Boyle Mark II (fig. 2).

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THE CONCENTRATION OF ANAESTHETICS IN CLOSED CIRCUITS WITH SPECIAL REFERENCE TO HALOTHANE

II. LABORATORY AND THEATRE INVESTIGATIONS

BY

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THE theoretical aspects of this problem have been dealt with by Mapleson (1960). The experiments in the laboratory and the observations in the operating theatre recorded in this paper were devised to check his conclusions and to lead the way to the clinical conclusions of Mushin and Galloon (1960).

The behaviour of the two types of closed circuit as defined by Mapleson was investigated. In the first, the patient's respirations pass through the vaporizer (vaporizer inside the breathing circuit): examples of this type are shown in figure 1, b–d. In the other, the vaporizer is in the fresh-gas supply line but outside the path of the patient's respirations (vaporizer outside the breathing circuit; figure 11).

VAPORIZER INSIDE THE BREATHING CIRCUIT

Laboratory Experiments

Method.

Three arrangements, of the vaporizer, the absorber and the fresh-gas input, commonly used in this country were investigated: the Boyle Mark II Circle (fig. 1, b), the Marrett Circle (fig. 1, c), and the M.I.E. Circle (fig. 1, d). All were standard models taken from the operating theatres and used without modification. Halothane was placed in the ether vaporizers. The expiratory valve at the face mask was used to allow any excess gases to leak off; all other leaks in the circuit were eliminated.

The pump representing the patient's respiration was an electrically-driven piston pump, with adjustable stroke and variable speed. This gave an approximately sine-wave pattern of respiration of adjustable tidal volume and rate.

The sampling and analysis circuit is shown in figure 1, a. A voltage stabilizer supplied a steady voltage to the katharometer, and a direct reading of halothane concentration was obtained on a galvanometer. A sampling flow through the analysis circuit was produced by inserting a resistance to air flow in the "trachea" (X in figure 1, b). The pressure drop across this produced a sampling flow through the katharometer during "inspiration". Flow during "expiration" was prevented by one-way valves (Y and Z). Soda lime was placed in the analysis circuit to absorb any carbon dioxide not removed by the absorber in the anaesthetic circuit, and also to keep constant the humidity of the vapour sample to the katharometer. The reference gas was oxygen.

At the beginning of each set of experiments, the zero was adjusted with oxygen passing through both sides of the katharometer. The galvanometer was then calibrated by passing oxygen via a sintered glass filter through halothane in the conical flask, so that halothane vapour saturated at a known temperature entered the analysis circuit. The concentration of this saturated vapour was calculated from a formula supplied by Messrs. I.C.I., Ltd.,* and the sensitivity of the galvanometer adjusted. This calibration was checked in the same way at the end of each set of experiments, usually 2 to 3 hours but not more than 5 hours from the start. The change in sensitivity was always found to be less than 5 per cent of the total concentration, and the readings were corrected on the assumption that the rate of change was constant. Because of this small change in sensitivity, the procedure was simplified for the subsequent laboratory experiments with the vaporizer out-

* $\log_{10} p = 11.7246 - \frac{2153.7}{T} - 0.006776T$ where p = pressure in mm Hg and T = absolute temperature.

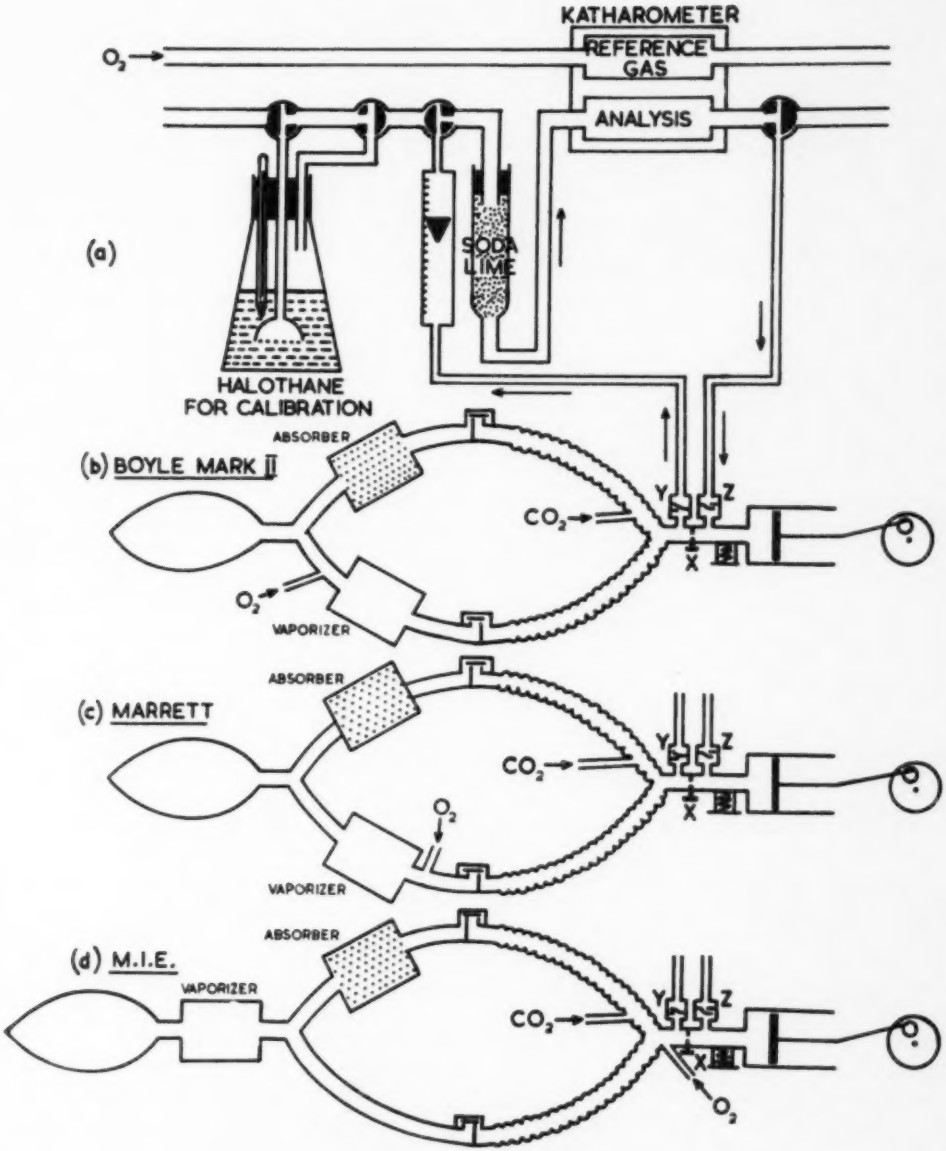

FIG. 1

Apparatus used in the laboratory for investigating the systems with the vaporizer inside the breathing circuit.

X: Resistance in "trachea" to produce sampling flow in "inspiration".
Y: One-way valve. Z: One-way valve.
(*a*) The analysis and sampling circuit.
(*b*) The Boyle Mark II Circle Absorber, with sampling leads and pump.
(*c*) The Marrett Circle Absorber (*d*) The M.I.E. Circle Absorber.

The M.I.E. Circle Absorber used throughout these investigations is the model prior to the recently introduced "Lincoln 800".

side the circuit, and all the observations in the operating theatre, by carrying out the calibration procedure only once at some time during each day. Zero drift was checked and adjusted half-hourly. The temperature of the laboratory was kept at $23 \pm 0.3^{\circ}C$ ($73.4 \pm 0.5^{\circ}F$).

The effect on "inspired" concentration of each of the following variables was investigated in turn:

1. Time from the start of the experiment.
2. Tap setting on the vaporizer.
3. Total fresh-gas flow into the circuit. Flows of 300 ml, 1000 ml and 3000 ml/min were used. A steady flow of 200 ml/min of carbon dioxide was added to the expiratory side of the anaesthetic circuit. This heated the soda lime in the absorber and hence the liquid in the vaporizer, thus reproducing conditions in the operating theatre. In addition, by its removal from the gases circulating in the circuit, the uptake of a comparable amount of oxygen by the patient was simulated. The rest of the total fresh-gas flow was made up with oxygen (100 ml, 800 ml and 2800 ml/min respectively).
4. Minute-volume ventilation:
   (*a*) 9 l./min (tidal volume 500 ml at a rate of 18/min).
   (*b*) 5.4 l./min (300 ml, 18/min).
   (*c*) 3.3 l./min (300 ml, 11/min).

Readings of the halothane concentrations were taken at 1-minute intervals for 20 minutes. At the end of each individual experiment the anaesthetic circuit was flushed with a high flow of oxygen to remove the halothane before starting the next experiment.

Results

Examples of the effect of changes in the above four variables on the "inspired" concentrations of halothane in the three circuits are shown in figures 2–5.

1. *Time.* In all the graphs there is an initial rapid rise in concentration which gradually flattens out. In most cases, within 20 minutes, the concentration has either risen above 10 per cent or has levelled out at some lower concentration.

Figure 2 shows that the curves for the M.I.E. are still rising fairly steadily after 20 minutes, whereas those for the Marrett and the Boyle Mark II are by that time almost flat. This is because in the M.I.E. the circulating gases have a very short path from the soda lime canister to the vaporizer (fig. 1, d), so that the heat from the canister gradually warms the liquid halothane in the vaporizer.

2. *Tap settings.* Figure 2 shows the effect on inspired concentrations of various positions of the control taps. As the tap is opened, the inspired concentration increases, but the increase is by no means proportional to the amount of movement of the tap, nor do the three taps provide the same range of concentrations for similar movements of the taps.

In figure 3 the concentrations obtained with different tap settings are related to the angle through which the control tap moves. This shows that the taps on these three vaporizers produce

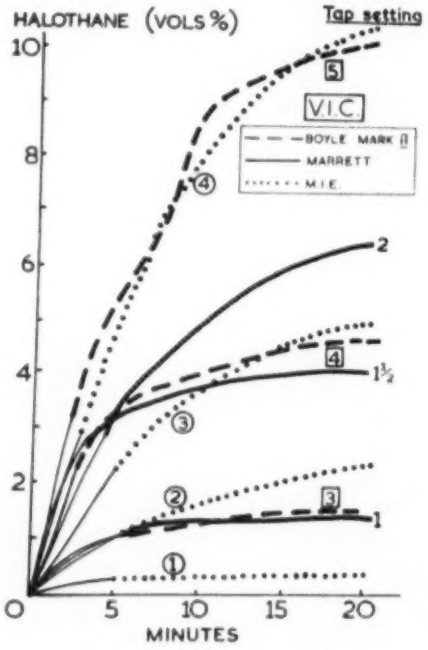

FIG. 2

Laboratory experiments (V.I.C.). The effect of various tap settings on the inspired concentration of halothane in the three vaporizers. Fresh-gas flow 1000 ml/min, ventilation 5.4 l./min in all cases. The numbers on the curves correspond to the tap settings.

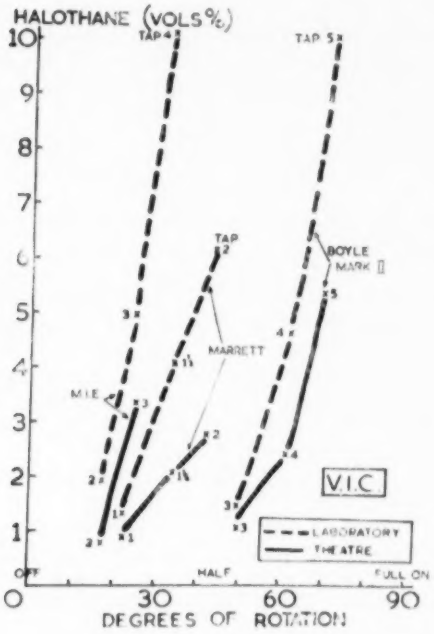

FIG. 3

The relation of the degree of rotation of the vaporizer taps to the inspired concentration of halothane, in the laboratory and in the operating theatre (V.I.C.). Fresh-gas flow 1000 ml/min both in the laboratory and in the operating theatre; ventilation 5.4 l./min in the laboratory, 4–6 l./min in the operating theatre.

marked increases in the inspired concentration with a small movement of the tap when an anaesthetic such as halothane is used.

It must be stressed here that these are laboratory experiments without a patient, where the factor of uptake by the patient does not exist. The concentrations of inspired halothane shown are therefore higher than those in the operating theatre, and, for comparison, figure 3 also shows concentrations found in the operating theatre for some of these tap settings, with a fresh-gas flow of 1000 ml/min and ventilation of 4–6 l./min.

3. *Fresh-gas input.* The point of entry of the fresh gas is different in the three circuits (fig. 1, b–d), but the effect of increasing the fresh-gas flow is always to reduce the concentration of halothane vapour inspired by the "patient" (fig. 4).

If the fresh-gas flow is zero, the gas in the breathing circuit recirculates indefinitely. Each time it passes through the vaporizer, it picks up more halothane vapour until eventually all the gas in the circuit reaches the saturation concentration of halothane at the temperature in the vaporizer. At room temperature this is approximately 35 per cent.

At the other extreme, if the fresh-gas flow is much larger than the minute-volume ventilation, the reservoir bag hardly fluctuates in size, and practically the whole of the patient's expirations therefore escapes through the expiratory valve. There is therefore little or no recirculation within the circuit and the patient inspires gas which has passed only once through the vaporizer (fig. 1, b) or has even reached him direct from the flowmeters (fig. 1, c, d).

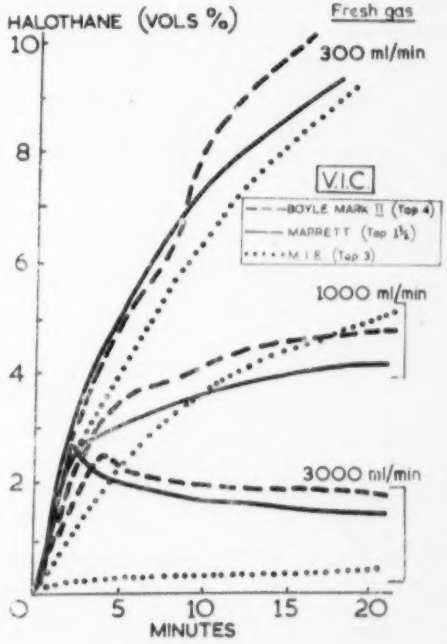

FIG. 4

Laboratory experiments (V.I.C.). The effect of various fresh-gas flows on the inspired concentration of halothane in the three vaporizers. Ventilation 5.4 l./min. The low curve for the 3000 ml/min flow in the M.I.E. is due to the position of the fresh-gas input in this circuit; at high flows much of the "inspiration" consists of fresh gas only.

Figure 4 shows a range of practical intermediate conditions.

The initial peaks in the curves for a 3 l./min fresh-gas flow in figure 4 are attributed to the vapour which accumulates between experiments above the liquid anaesthetic inside the vaporizer, and is released into the circuit when the vaporizer is first turned on (Mapleson, 1957). At lower fresh-gas flows the peak is swamped by the rapid rise in concentration.

4. *Minute-volume ventilation.* In these anaesthetic circuits the vaporizer is within the anaesthetic system. Vaporization is effected by the patient's ventilation. Therefore if the ventilation increases, the rate of vaporization increases. Vapour begins to accumulate in the circuit and the circuit concentration begins to rise. However, as it rises, and if the fresh-gas input is above basal flows, so the rate of loss of vapour through the expiratory valve also rises, until the rate of loss again equals the rate of vaporization. Equilibrium is re-established at a higher inspired concentration. The increase of inspired concentration with an increase of ventilation is illustrated in figure 5.

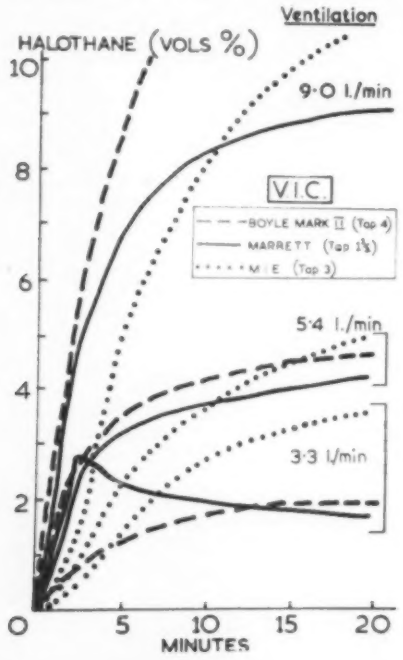

FIG. 5

Laboratory experiments (V.I.C.). The effect of various minute-volume ventilations on the inspired concentration of halothane in the three vaporizers. Fresh-gas flow 1000 ml/min.

Observations made in the Operating Theatre (V.I.C.)

The apparatus was the same as that used in the laboratory, except, of course, that carbon dioxide from a cylinder was not added. A continuous sample was taken from the inspiratory side of the breathing circuit by means of a pump set to produce a sampling flow of 800 ml/min, since the resistance used in the laboratory to drive a sample to the katharometer could not be used in the theatre. The sample was returned to the inspiratory side just proximal to the patient.

Anaesthetic method.

Premedication was with morphine and atropine in appropriate dosage, with an additional 0.6 mg atropine intravenously just prior to induction of anaesthesia. Thiopentone (200–400 mg) was followed by suxamethonium (30–50 mg). The vocal cords were sprayed with 4 per cent lignocaine, a cuffed endotracheal tube was inserted and connected to the breathing circuit. In all cases breathing was spontaneous.

A high flow of oxygen was used until the galvanometer reading was zero, thus indicating that the air in the apparatus and nitrogen from the patient had been eliminated from the circuit. This was found to take 4 to 5 minutes, and thereafter the oxygen was turned down to the chosen fresh-gas flow and the vaporizer tap opened.

Results.

Figures 6–10 are typical of the records obtained from a number of patients, using the same three circle absorbers as in the laboratory experiments. In each case, the inspired concentrations of halothane are plotted against time in minutes after the opening of the vaporizer tap. The minute-volume ventilation figures (in l./min) were obtained by taking readings at 1-minute intervals from a Dräger Volumeter placed in the inspiratory side of the circuit; these actual readings were corrected for the inaccuracies in the Volumeter which occur at low ventilations, and the final

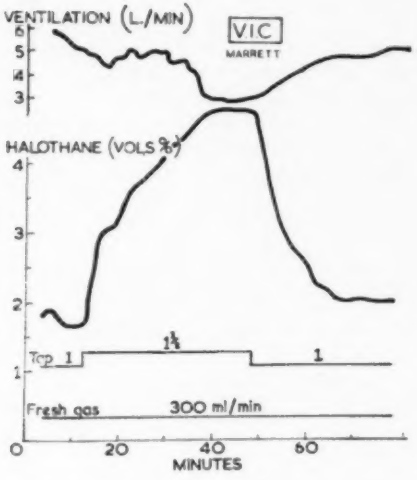

FIG. 6
Theatre observations (V.I.C.). Inspired concentrations of halothane obtained with a Marrett vaporizer, showing the effect of changes in the tap setting, with a fresh-gas flow of 300 ml/min.

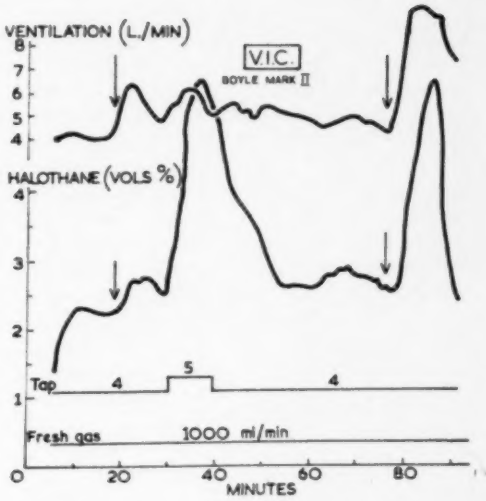

FIG. 8
Theatre observations (V.I.C.). Inspired concentrations of halothane obtained with a Boyle Mark II vaporizer, showing the effect of changes in the tap setting and in the ventilation with a fresh-gas flow of 1000 ml/min.

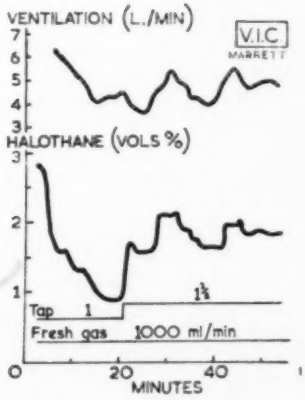

FIG. 7
Theatre observations (V.I.C.). Inspired concentrations of halothane obtained with a Marrett vaporizer, showing the effect of changes in the tap setting, with a fresh-gas flow of 1000 ml/min.

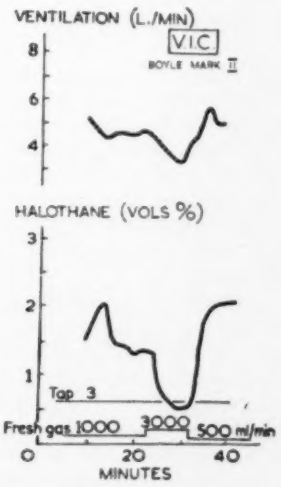

FIG. 9
Theatre observations (V.I.C.). Inspired concentrations of halothane obtained with a Boyle Mark II vaporizer, showing the effect of changes in the fresh-gas flow.

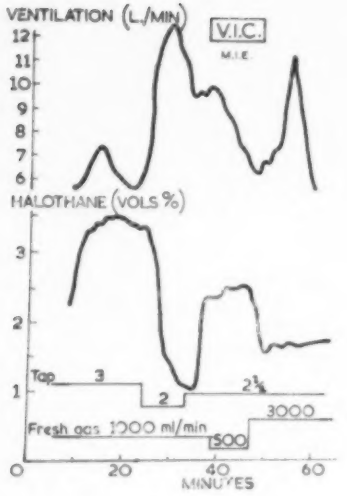

FIG. 10

Theatre observations (V.I.C.). Inspired concentrations of halothane obtained with an M.I.E. vaporizer, showing the effect of changes in the tap setting and the fresh-gas flow. The ventilation in this patient was higher than in the others, as morphine was not given pre-operatively.

figures shown in the graphs were obtained by successive 5-minute averages of the corrected readings.

Figures 6–10 show the concentration of halothane inspired by the patient at various tap settings, and illustrate the narrow range of movement of the taps covering the whole of the clinical range of concentrations of halothane.

The effect of changes in the fresh-gas flow is shown in figures 9 and 10; the increase in inspired concentration with a reduction in the fresh-gas flow is well shown.

The effect of changes in ventilation on the inspired concentration is seen in figure 8. At the first arrow, a spontaneous increase in ventilation occurred as a result of the stimulus of the start of the operation, and this increased the concentration from 2.2 to 2.8 per cent; when the ventilation fell again, the concentration also began to fall. At the second arrow, the bag was rhythmically squeezed, increasing the ventilation from about 4.5 l./min to 10 l./min; the very steep rise in inspired concentration following this manoeuvre speaks for itself.

The laboratory experiments showed that an increase in ventilation increases the vaporization and therefore the inspired concentration. In the theatre an increase in ventilation similarly causes an increase in the inspired concentration, and therefore in the alveolar concentration; anaesthesia becomes deeper as a result. In addition to these events, the increased ventilation produces an increase in the transfer of anaesthetic to the alveoli, so that the alveolar concentration approaches more nearly to the inspired concentration. This accentuates the effect of ventilation on the depth of anaesthesia.

Because of the higher uptake of anaesthetic by the patient which occurs with higher alveolar concentrations, the measured rise in *inspired* concentration with an increase in ventilation is not as great as in the laboratory, where there is no uptake.

Conclusions (Vaporizer inside the Breathing Circuit)

1. *Time.* In both the laboratory and the theatre, the inspired concentration increases very rapidly at first, but within 10 minutes begins to flatten out, and in most cases reaches a steady level by 20 minutes. A progressive "build-up" of inspired vapour concentration does not occur even in the laboratory unless a truly closed circuit with basal fresh-gas flows is used, when it builds up towards the saturation concentration of halothane (35 per cent at room temperature). With higher fresh-gas flows the inspired concentration reaches a steady level at some level lower than the saturation concentration within 10–20 minutes.

In the operating theatre, *even with basal fresh-gas flows*, a steady level is reached in the same time. Halothane is being taken out of the circuit by the patient, and therefore equilibrium is reached between the amount of halothane added to the circuit from the vaporizer, and the amount being taken up by the patient. A "build-up" of inspired concentration could only occur when the patient is saturated with halothane, and therefore not removing any from the circuit. Experiments on the uptake of halothane (Duncan and Raventós, 1959) indicate that saturation of the body would only be reached after many hours; similar observations on other anaesthetics

show that the same is true for them (Haggard, 1924; Severinghaus, 1954).

The magnitude of this steady level depends on the several factors described in this paper. It does not necessarily fall within the acceptable clinical range (Mushin and Galloon, 1960).

2. *Tap setting.* An increase in the tap setting increases the inspired concentrations. In the laboratory, the different makes of vaporizer all show the same restricted range of control over the inspired concentrations; in the theatre, however, there appears to be some difference in the fineness of the control in the different vaporizers (see fig. 3). Two vaporizers of the same make may also produce slightly different inspired concentrations at the same tap setting.

3. *Fresh-gas flow.* An increase in the fresh-gas flow reduces the inspired concentration. This can be regarded as due to a dilution of the anaesthetic vapour in the circuit by the higher fresh-gas flow, or to a greater rate of escape of anaesthetic vapour through the expiratory valve; this must necessarily occur at fresh-gas flows greater than basal.

4. *Minute-volume ventilation.* An increase in the minute-volume ventilation increases the vaporization of the anaesthetic and therefore the inspired concentration.

5. *Absorption of anaesthetic by rubber.* A sharp initial peak of high concentration can be seen in figures 7 and 9. This has already been referred to in the laboratory experiments, and has also been reported with trichloroethylene vaporization (Mapleson, 1957). The high rate of absorption of trichloroethylene by the rubber in the anaesthetic circuit prevents this initial high concentration reaching the patient. The absorption of halothane vapour by rubber is, however, much lower, so that the initial high concentration is delivered to the patient. Since the circuit is closed, it is some minutes before this peak is dissipated. This effect is particularly well shown with the Marrett because the volume above the liquid in the vaporizer is the largest of the three.

VAPORIZER OUTSIDE THE BREATHING CIRCUIT

Laboratory Experiments

Method.

The absorption circuit used was a Waters to-and-fro system; the vaporizer was a standard Boyle trichloroethylene bottle containing 75 ml of halothane (fig. 11). The fresh gases (oxygen and carbon dioxide) were passed through this bottle and entered the circuit close to the expiratory valve. Before starting the experiments, the Boyle bottle was calibrated in terms of the per-

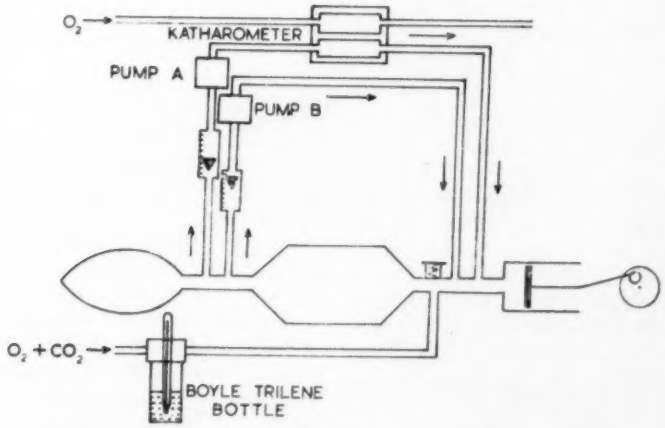

FIG. 11

Apparatus used in the laboratory for investigating the system with the vaporizer outside the breathing circuit (V.O.C.).

centage saturation of the issuing vapour for a range of tap settings and fresh-gas flows, the plunger being kept at a constant distance of 7 mm above the surface of the liquid. The concentration obtained at any time could then be estimated from the calibration and a knowledge of the tap setting, fresh-gas flow, temperature of the liquid halothane and atmospheric pressure, on the assumption that at any particular tap setting and flow rate, the vaporizer always delivers vapour at the same degree of saturation.

A small pump (A), set at a flow of 800 ml/min, delivered a continuous sample to the katharometer from a point between the reservoir bag and the Waters canister; the sample was returned to the patient side of the expiratory valve. The second pump (B), set at a flow of 3000 ml/min, ensured thorough mixing of the gases and vapours in the circuit.*

The rest of the apparatus and the procedure was as already described for the vaporizer inside the breathing circuit, and again the effect of the following variables was determined:

1. Time from the start of the experiment.
2. Tap setting (i.e. different input concentrations).
3. Fresh-gas flow: as before, 200 ml/min of carbon dioxide was added to the circuit, and made up with oxygen to total fresh-gas flows of 500 ml, 1000 ml and 3000 ml/min.
4. Minute-volume ventilation: 9, 5.4 and 3.3 l./min.

Results.

The effect of these four variables on the "inspired" concentration is shown in figures 12–15. The input concentration referred to in the graphs is the concentration of halothane in the fresh gases delivered to the circuit close to the expiratory valve.

1. *Time.* Figure 12 shows the inspired concontrations of halothane with two different input concentrations, the fresh-gas flow being 1000 ml/min in each case. In the absence of uptake of halothane by the patient, the inspired concentration increases rapidly at first, and then more and more slowly; within 20 minutes, the inspired concentration has settled at a steady level which is above that of the input concentration.

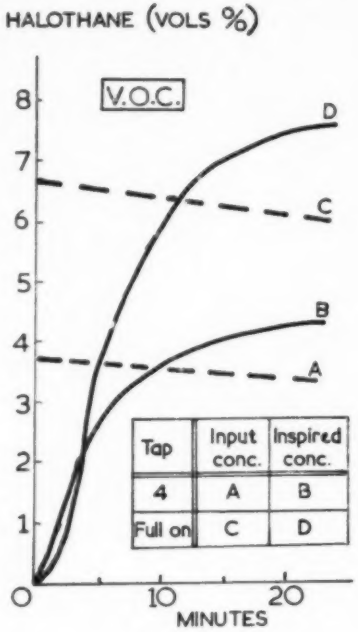

FIG. 12

Laboratory experiments (V.O.C.). The effect of two different input concentrations on the inspired concentration of halothane. Fresh-gas flow 1000 ml/min, ventilation 5.4 l./min in both. The input concentration is the concentration of halothane in the fresh gases leaving the vaporizer and entering the circuit close to the expiratory valve. The gradual fall in the input concentration is due to cooling of the halothane in the Boyle bottle.

A source of variation in the results was found to be the absorption of halothane by the rubber in the anaesthetic circuit. In the first experiment of any day, the inspired concentration increased rather more slowly than in a similar experiment after the rubber had been exposed to halothane for some time and had, presumably, become saturated. Because of this, the graphs shown in figures 12 and 13 refer to experiments where the rubber had become saturated with halothane, whilst those

* The method of sampling differed from that used in the laboratory experiments with the vaporizer inside the breathing circuit, because by this time the method of continuous sampling had been evolved for use in the operating theatre. It was felt that any errors introduced by using this in the laboratory with the vaporizer out of the breathing circuit would be small (certainly less than 5 per cent of the input concentration) and, of course, systematic.

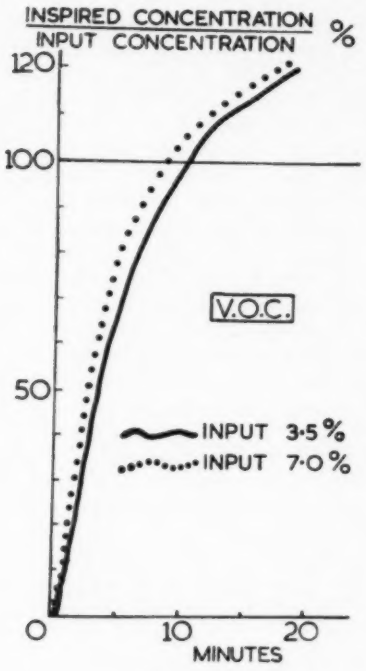

FIG. 13

Laboratory experiments (V.O.C.). The effect of two different input concentrations on the inspired concentration of halothane, expressed as a percentage of the input concentration. Fresh-gas flow 1000 ml/min, ventilation 5.4 l./min.

in figures 14 and 15 refer to experiments performed when the rubber had not been exposed to halothane for some hours. A comparison of figure 13 with the middle graph of figure 14 (5.4 l./min, fresh-gas flow 1000 ml/min), shows that this effect is quite appreciable.

2. *Tap settings.* If the inspired concentrations shown in figure 12 are expressed as a percentage of the corresponding input concentrations (fig. 13), it can be seen that the rate of increase of inspired concentration, and the final steady level reached, are both independent of the input concentration (tap setting), at least over the range 3.5 per cent to 7 per cent.

3. *Fresh-gas flow.* The wide range of fresh-gas flows used in this part of the experiment produces appreciable differences in the concentration of halothane leaving a vaporizer of the Boyle trichloroethylene bottle type. The effect of the fresh-gas flow is therefore more clearly shown by expressing the inspired concentration as a percentage of the input concentration. Figure 14 shows that the higher the fresh-gas flow, the more rapidly does the inspired concentration increase, but the lower is the final steady level.

The explanation for this is as follows. At the start of the experiment, the circuit concentration is low, so that loss of anaesthetic through the expiratory valve is negligible. The rate of increase of concentration in the circuit is therefore proportional to the rate of input, and therefore to the fresh-gas flow. For example, 500 ml/min of 5 per cent halothane in oxygen will carry 25 ml/min of halothane vapour into the circuit, whilst 3000 ml/min of 5 per cent halothane in oxygen will carry 150 ml/min of halothane vapour.

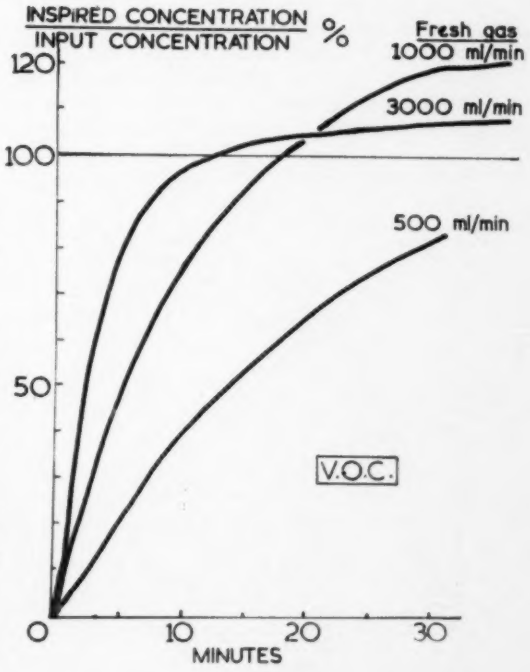

FIG. 14

Laboratory experiments (V.O.C.). The effect of various fresh-gas flows on the inspired concentration of halothane, expressed as a percentage of the input concentration. Ventilation 5.4 l./min.

However, as the circuit concentration increases, the loss of vapour through the expiratory valve becomes appreciable. Eventually (usually within 20 minutes), a steady level will be reached in which the rate of loss of anaesthetic from the circuit is equal to the rate of input.

Figure 14 shows that this steady level also depends on the total fresh-gas flow. If this is very large, the circuit is well flushed with fresh gas; the removal of the 200 ml/min of carbon dioxide (simulating the oxygen uptake of the patient) is a negligible part of the fresh-gas flow, so that the inspired concentration equals the input concentration (the circuit is therefore in effect a non-rebreathing one). If the fresh-gas flow is low, the simulated uptake of oxygen represents an appreciable fraction of the input flow, and halothane is left to "accumulate" in the circuit. The lower the fresh-gas flow, therefore, the higher is the final steady level.

If the fresh-gas flow is 1000 ml/min, the final level of the inspired concentration is about 125 per cent of the input concentration, and if it is 3000 ml/min, the final level is about 107 per cent. A brief theoretical consideration gives the reasons for these laboratory figures. If the input flow is 1000 ml/min at, say, 4 per cent halothane, this contains 40 ml/min of halothane vapour. Two hundred ml/min of this flow consists of the added carbon dioxide, which is absorbed by the soda lime. The loss through the expiratory valve is therefore 800 ml/min, which, when the steady level is reached, must contain 40 ml/min of halothane vapour; the loss is therefore at a concentration of 5 per cent. This concentration must be the concentration in the circuit, or the inspired concentration, which is therefore 125 per cent of the input concentration. Similar calculations for fresh-gas flows of 500 and 3000 ml/min show that the final steady levels are 166 per cent and 107 per cent, respectively, of the input concentration.

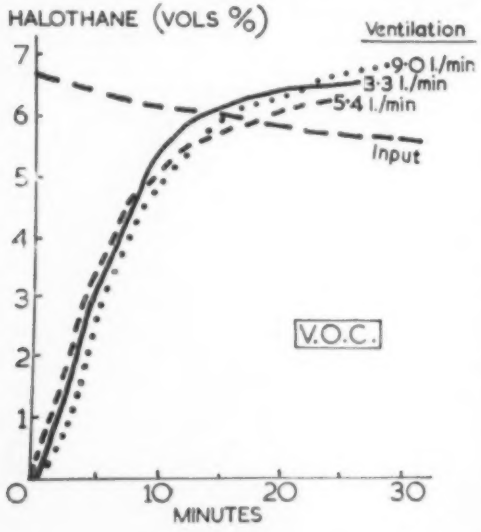

FIG. 15

Laboratory experiments (V.O.C.). The effect of various minute-volume ventilations on the inspired concentration of halothane. Fresh-gas flow 1000 ml/min.

4. *Minute-volume ventilation.* Variations in ventilation would only affect the inspired concentration if the patient extracted halothane from the circuit. Figure 15 shows that in the laboratory the rate of increase of inspired concentration and the final steady level reached are not affected by changes in minute-volume ventilation.

Observations made in the Operating Theatre (V.O.C.)

Method.

The apparatus used was again the same as that in the laboratory experiments with the vaporizer outside the breathing circuit—a Waters to-and-fro absorption system, with oxygen passing through a standard Boyle trichloroethylene bottle containing 75 ml of halothane; the oxygen and halothane entered the circuit near the expiratory valve close to the patient. The sampling and mixing arrangements were also the same as in the laboratory.

Induction of anaesthesia was the same as that described in the section on theatre observations with the vaporizer inside the breathing circuit.

Results.

Figures 16–18 show the effect on the inspired concentration in three patients of changes in the tap setting, fresh-gas flow, and minute-volume ventilation.

1. *Tap setting.* In figure 18, an input of 4¼ per cent halothane with 500 ml/min of oxygen produces an inspired concentration of about 2

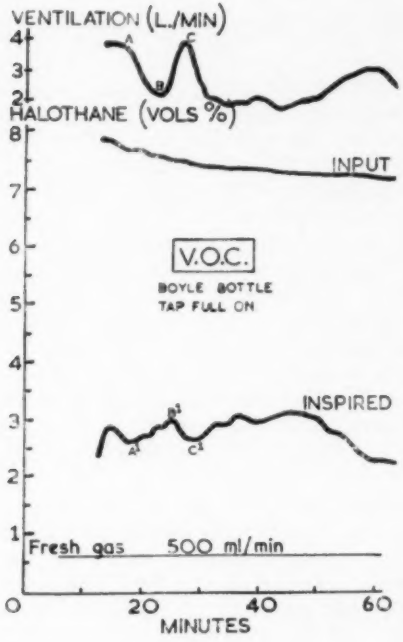

FIG. 16

Theatre observations (V.O.C.). Input and inspired concentrations of halothane obtained with a Boyle trichloroethylene bottle leading into a to-and-fro absorption system, showing the effect of changes in ventilation. Fresh-gas flow 500 ml/min. Tap setting full on, plunger just above the surface of the liquid.

per cent, whilst in figure 16 an input of 7½ per cent produces an inspired concentration of 3 per cent. In the laboratory experiments, whatever the input concentration, the inspired concentration settled to a steady level which was in all cases above the input concentration. In the theatre the important difference is that the patient is removing anaesthetic from the circuit, and the final steady level is reached in about 10 minutes when the rate of uptake of anaesthetic by the patient equals the rate of input from the vaporizer.

It can be seen from the figures that this steady level of inspired concentration is below that of the input concentration, and it will remain so as long as the patient continues removing anaesthetic from the circuit.

2. *Fresh-gas flow.* Figure 17 shows that a decrease in the fresh-gas flow from 1000 to 500 ml/min (with the same tap setting) increases the concentration of halothane leaving the Boyle bottle. In spite of this higher input, however, the inspired concentration falls from about 4 per cent to 2½ per cent. This is in contrast to the laboratory experiments, where lower fresh-gas flows produced higher "inspired' concentrations. In the operating theatre, halothane is taken up by the patient more rapidly than oxygen in relation to its concentration; therefore it is now the oxygen that "accumulates" in the circuit. This effect is most marked at low fresh-gas flows; at high flows the circuit is well flushed and the effect is negligible.

3. *Minute-volume ventilation.* The effect of changes in ventilation can be seen in figure 16. Here a fall in ventilation (from A to B) produces

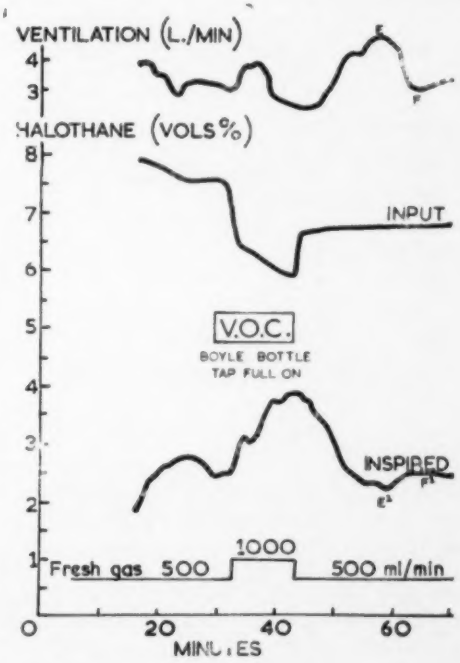

FIG. 17

Theatre observations (V.O.C.). Input and inspired concentrations of halothane obtained with a Boyle trichloroethylene bottle leading into a to-and-fro absorption system, showing the effect of changes in the fresh-gas flow. Tap setting full on, plunger just above the surface of the liquid.

a rise in inspired concentration (A^1 to B^1), whilst an increase in ventilation (from B to C) produces a fall in inspired concentration (B^1 to C^1). Similar effects are shown at points E and F in figure 17, and throughout figure 18.

With the vaporizer outside the breathing circuit, changes in minute-volume ventilation have no effect on the rate of vaporization of the anaesthetic. An increase in ventilation produces an improved transfer of anaesthetic to the alveoli, so that there is a smaller difference between the circuit (inspired) and alveolar concentrations. In general this appears partly as a rise in the alveolar concentration (and hence an increase in the rate of uptake of anaesthetic by the patient), and partly as a fall in the inspired concentration (due to the increase in the rate of uptake) (Mapleson, 1960).

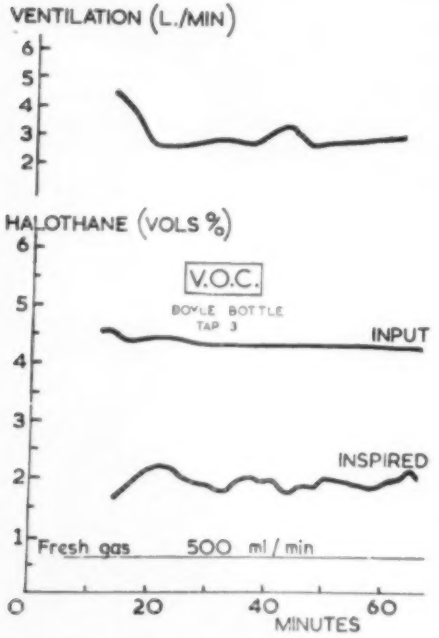

FIG. 18

Theatre observations (V.O.C.). Input and inspired concentrations of halothane obtained with a Boyle trichloroethylene bottle leading into a to-and-fro absorption system, showing the effect of a lower input concentration. Tap setting ¾ mark, plunger just above the surface of the liquid. Fresh-gas flow 500 ml/min.

Conclusions (Vaporizer outside the Breathing Circuit)

1. *Time.* A progressive "build-up" of inspired concentration does not occur in these circumstances for the same reasons as in the case of the vaporizer inside the breathing circuit. From figures 16–18 it is clear that a steady level of inspired concentration is reached within 10–20 minutes, even with very low flows.

2. *Tap setting.* Changes in the input concentration (tap setting) produce proportional changes in the rate of increase of inspired concentration, and in the final steady level reached. In the laboratory this steady level is in all cases above the input concentration; in the theatre it is below the input concentration as long as the patient continues extracting halothane from the circuit.

3. *Fresh-gas flow.* In the laboratory, the lower the fresh-gas flow, the slower is the initial increase in the inspired concentration, but the higher is the final steady level reached. In the theatre, the lower the fresh-gas flow, the lower is the inspired concentration, despite any increase in input concentration that may occur in simple vaporizers like the Boyle bottle when the fresh-gas flow is reduced.

4. *Minute-volume ventilation.* An increase in ventilation reduces the inspired concentration in the operating theatre, and vice versa, unlike the laboratory, where changes in ventilation have no effect on the inspired concentration.

SUMMARY

The effect, on the concentration of halothane inspired, of variations in time, tap setting, fresh-gas flow, and minute-volume ventilation has been studied in the laboratory and in the operating theatre for the two types of closed circuit. Table I summarizes the effect on the inspired concentration of changes in these factors *in the operating theatre.*

Although the effects of the above changes have been described throughout in terms of halothane concentration, the general principles outlined and the conclusions drawn apply to the vaporization of any liquid anaesthetic with a high enough rate of uptake in these types of anaesthetic systems.

TABLE I

	Vaporizer inside the breathing circuit	Vaporizer outside the breathing circuit
Increase in tap setting	Increased	Increased
Increase in fresh-gas flow	Decreased	Increased
Increase in ventilation	Increased	Decreased*
Time	Equilibrium reached in 10–20 minutes, at a level determined by the other factors. No progressive "build-up" even with basal fresh-gas flows.	

* Although the inspired concentration is decreased, the alveolar concentration, and hence the depth of anaesthesia, is increased (see text and Mushin and Galloon (1960))

ACKNOWLEDGMENTS

A practical study of this extent necessarily involves the help of many colleagues. In particular, I would like to thank Professor William W. Mushin for starting me on it, and for his wise advice through all its stages; the completion of the work is in no small measure due to his encouragement. My thanks are due also to W. W. Mapleson, Ph.D., for his constant scientific guidance; his developing theoretical studies constantly stimulated my experimental ones. I would like to thank Mr. E. K. Hillard, Senior Technician to this Department for his technical help, always willingly given; and all the senior and junior members of the Department who helped, directly or indirectly, to complete the investigation. In addition, my thanks are due to my surgical colleagues for their patience and forbearance during the observations made in the operating theatre.

Lastly, but not least, my grateful thanks to the Directors of Imperial Chemical Industries (Pharmaceuticals) Ltd., for liberal supplies of Fluothane for the many experiments described here; and to their scientific staff for helpful data.

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BOOK REVIEW

General Anaesthesia for Dental Surgery. By R. S. Walsh, M.A., B.M., B.CH., F.F.A.R.C.S., First Assistant, Department of Anaesthetics, St. George's Hospital, and the Southampton Group of Hospitals. Published by Longmans, London. Pp. 94. Price 21*s*.

It is a pleasure to welcome this essentially practical work on general anaesthesia for dentistry. Its publication at a time when the sequels, both potential and real, to anaesthesia in the dental chair are undergoing appraisal is fortunate, and will certainly add to its value. The subject matter is not provocative, being based on the use of nitrous oxide, but makes the sort of companion to clinical practice that every medical and dental student should read during undergraduate training, and then again after qualification if the prospect of giving a dental "gas" seems imminent. There is sufficient emphasis on the potential dangers of hypoxia to deter all but the foolhardy from the temptation of untutored practice, and a collection of useful pictures to illustrate the finer arts of maintaining anaesthesia in the dental chair while supporting the jaw for the dental surgeon.

W. D. Wylie

Brit. J. Anaesth. (1960), **32**, 324

THE CONCENTRATION OF ANAESTHETICS IN CLOSED CIRCUITS WITH SPECIAL REFERENCE TO HALOTHANE

III. CLINICAL ASPECTS

BY

WILLIAM W. MUSHIN AND S. GALLOON

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THE factors which influence the concentration of volatile anaesthetics in a closed circuit have been studied theoretically by Mapleson (1960) and experimentally by Galloon (1960). This paper draws conclusions from their findings and relates them to the circumstances of clinical anaesthesia in the operating theatre. The various aspects of the problem are presented in terms of halothane, but the conclusions reached could in general be applied equally to other volatile anaesthetic agents when used in a closed circuit.

ECONOMY

If the fresh gas flow into an anaesthetic circuit is greater than the basal oxygen requirement of the patient, some gases must escape to the atmosphere through the expiratory valve and carry anaesthetic vapour with them. Therefore, whether the vaporizer is inside or outside the breathing circuit, the wastage of vapour is governed by the fresh-gas flow and the concentration of the vapour.

Thus, in unit time,

Volume of gases escaping from circuit	=	Volume of fresh gas entering circuit − Volume of oxygen removed by patient
But, concentration of anaesthetic vapour in escaping gases	=	Concentration of anaesthetic vapour in circuit
Therefore, loss of anaesthetic vapour	=	Concentration of anaesthetic vapour in circuit × (Volume of fresh gas supplied − Volume of oxygen removed by patient).

Figure 1* shows the relationship between the wastage of liquid halothane and fresh-gas flow.

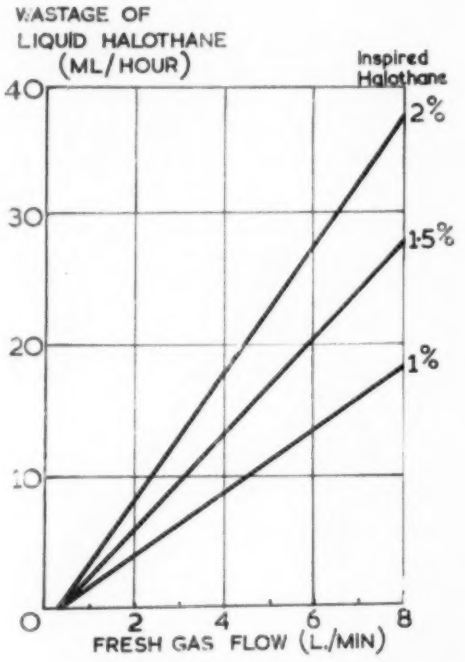

FIG. 1

The wastage of liquid halothane related to the fresh-gas flow into the circuit (V.I.C. and V.O.C.). The uptake of halothane by the patient has not been included in this graph, which shows wastage, and not total usage.

* The values shown in figure 1 are modified if the fresh-gas flow enters the circuit close to the expiratory valve. In the case of the V.I.C. (e.g. the M.I.E.) the wastage of anaesthetic vapour with fresh-gas flows over 3 l./min is rather less than shown in figure 1, because some of the fresh gas escapes directly through the expiratory valve; with smaller flows the difference between this and other circuits becomes negligible. In the case of the V.O.C. some of the loss through the valve is in the form of a vapour concentration direct from the vaporizer and the wastage is therefore somewhat higher than shown, especially at high fresh-gas flows.

With a basal oxygen supply alone, there is no wastage, and it is only with fresh-gas flows above 1000 ml/min that the wastage becomes of importance.

With the vaporizer inside the breathing circuit (V.I.C.)† a reduction in the fresh-gas flow leads to an increase in the inspired concentration for any particular vaporizer tap setting; figure 2 shows that in spite of this, a reduction in fresh gas always results in economy. It follows from this graph that if economy is obtained by turning down the fresh-gas flow, it will also be necessary to use lower tap settings on the vaporizer in order to obtain the same inspired concentrations. Therefore in the V.I.C. the greatest economy is only obtained at the price of restricting the useful range of tap settings and hence of a greater difficulty in fine control of the inspired concentration.

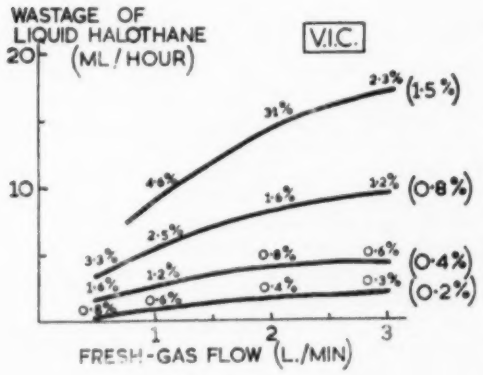

FIG. 2

The effect of the fresh-gas flow on the wastage of liquid halothane and on the inspired concentration in the V.I.C. The figures above each curve give the inspired concentrations at the particular fresh-gas flow. Each curve represents a different tap setting, or vaporizer concentration (the figures in brackets on the right of each curve). If the fresh gas is reduced, the wastage falls, but the inspired concentration rises.

With the V.O.C. a reduction in the fresh-gas flow leads to a reduction in the inspired concentration; figure 4 shows that if very small fresh-gas flows are used for reasons of economy, the vaporizer in the V.O.C. must be capable of delivering high concentrations.

The lower limit of fresh-gas flow is generally dictated by the use of nitrous oxide.

If x = O_2 flow in ml/min
and y = N_2O flow in ml/min
Then total gas flow = $(x+y)$ ml/min

If the basal O_2 requirement is assumed to be 200 ml/min, then if the average concentration of O_2 is to be 20 per cent, it can be shown that

$$\frac{20}{100} = \frac{x-200}{x+y-200}^{*}$$

$$800 = 4x - y$$

$$200 + \frac{y}{4} = x$$

i.e. if $x = 200$ ml, *NO* N_2O can be used.

In the N_2O flowmeters commonly supplied with anaesthetic apparatus the smallest flow-rate which can be measured conveniently is 1000 ml/min. This means that for a 20 per cent O_2 concentration the volumes are O_2 450 ml/min, and N_2O 1000 ml/min. For a 50 per cent O_2 concentration, it can be shown that the O_2 flow must be 1200 ml/min if the N_2O is 1000 ml/min. As soon therefore as N_2O is introduced, the minimum practical flow of fresh gas is between 1500 and 2500 ml/min (fig. 3).

It follows, therefore, that if nitrous oxide is used the minimum wastage of halothane is much higher than can be achieved with oxygen alone.

The curves shown in figure 3 were based on the above formula. This is a broad approximation only and caution should be exercised in transferring the data to clinical practice if low oxygen flow rates are used. The curves are based on the assumption that the oxygen utilization is 200 ml/min. The actual utilization may vary widely from this figure, with corresponding variation in the inspired oxygen concentration.

CONTROL OF INSPIRED CONCENTRATION

It is clearly desirable that the inspired concentration of anaesthetic should be easily controlled

†The contractions V.I.C. and V.O.C. signify "vaporizer inside the breathing circuit" and "vaporizer outside the breathing circuit" respectively.

* In fact this is the concentration of O_2 leaving the circuit through the expiratory valve when the uptake of N_2O is neglected. The actual inspired concentration of O_2 would be a little higher.

by the anaesthetist, and that in general the effect of circumstances beyond his control should be small.

The principal factors influencing the inspired concentrations are the "vaporizer concentration"* (i.e. the tap setting), the fresh-gas flow, the ventilation, and the rate of uptake of anaesthetic from the lungs into the blood.

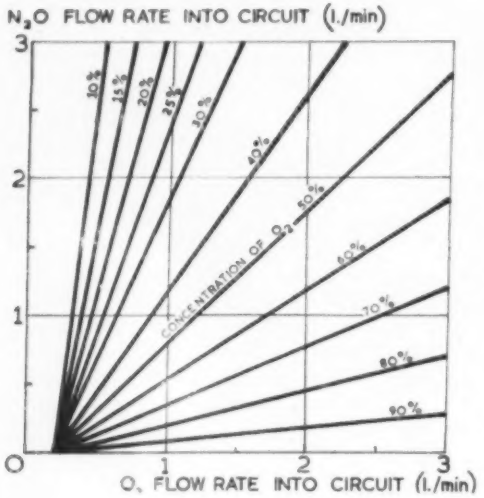

FIG. 3

The relation between the concentration of the gases in an anaesthetic circuit and the flow rates of oxygen and nitrous oxide into it. The uptake of oxygen by the patient is assumed to be 200 ml/min. The uptake of nitrous oxide is neglected. For derivation see text.

(a) *Vaporizer concentration.*

Ideally a vaporizer for use in the closed circuit should have the following attributes:

1. The vaporizer concentration should be known.
2. The vaporizer concentration should be constant for each tap setting, and should be independent of variations in gas flow through it.
3. A vaporizer for use in the V.I.C. should have a low efficiency, whilst one for use in the V.O.C. must have a high efficiency.
4. The movement of the control tap should allow the anaesthetist fine control over the concentration delivered to the patient.
5. The air space above the liquid anaesthetic in the vaporizer should be small.

Of the many different vaporizers in use for halothane in the closed circuit in this country, only a few (e.g. the Fluotec Mark II and the modified Rowbotham) can be said to have been designed for this specific purpose, and only the Fluotec comes near to satisfying all the above attributes.

No figures are available at present which give the vaporizer concentration for any of the vaporizers in use inside the breathing circuit. With the vaporizer outside the breathing circuit, the vaporizer concentration may be known to the anaesthetist if the vaporizer has been calibrated. With both arrangements, the vaporizer concentration, in the case of available vaporizers, is subject to variations with changes in flow and temperature, and any calibration must have reference to at least these two factors.

Consideration of the other two papers in this series confirms that the vaporizer concentration in the V.I.C. for an anaesthetic such as halothane should not exceed a range of up to 2 per cent (fig. 4). A vaporizer of even lower efficiency is adequate in a circuit where the patient's respirations pass to-and-fro through the vaporizer, such as the M.I.E. circle absorber (Mapleson, 1960; fig. 2). Similarly, a vaporizer for use outside the breathing circuit should be capable of delivering concentrations up to at least 6 per cent (fig. 4). The common practice of using a Fluotec Mark II with small fresh-gas flows and a to-and-fro absorption system may appear to contradict this latter statement, but with small fresh-gas flows the concentrations delivered by this vaporizer are generally higher than those indicated on the dial (e.g. with a fresh-gas flow of 500 ml/min and the dial set at 3 per cent, the concentration delivered is 4 per cent whilst with a dial setting of 4 per cent the concentration is 7 per cent).

The relationship of movement of the control tap of the vaporizer to alterations in the vaporizer concentration should allow the anaesthetist fine

* This term is fully discussed in the first paper in this series (Mapleson, 1960). In the case of the V.O.C., the vaporizer concentration is the same as the concentration leaving the vaporizer and entering the circuit. In the case of the V.I.C., the vaporizer concentration is approximately the amount by which the concentration is increased as gas passes through the vaporizer.

control over the concentration delivered to the patient. With the exception of the Fluotec, most of the vaporizers available for closed circuit halothane were designed for ether, and a small movement of the vaporizer tap produces a marked difference in inspired concentration when an anaesthetic such as halothane replaces the ether (fig. 5).

Another important consideration is the volume of the space above the liquid in the vaporizer, in which a saturated vapour collects between cases. If this volume is large, then when the vaporizer tap is first opened, a considerable quantity of concentrated vapour enters the circuit to be inspired by the patient for the first few minutes. A sudden high concentration inspired during induction, may be more dangerous than the same concentration produced more gradually. The space above the liquid in the vaporizer bottle should therefore be as small as possible. This applies whether the vaporizer is inside or outside the breathing circuit.

(b) *Fresh-gas flow.*

The fresh-gas flow is directly under the control of the anaesthetist. The greatest economy of

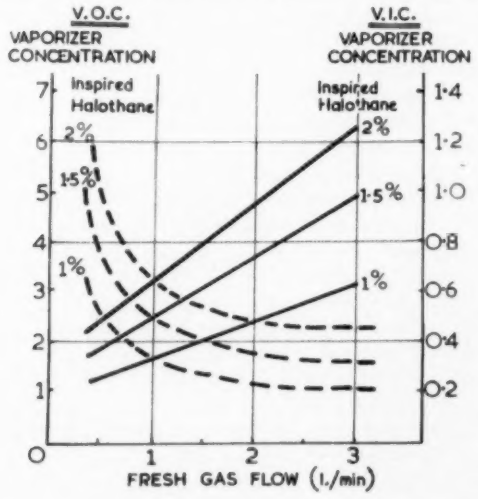

FIG. 4

Vaporizer concentrations required to obtain inspired concentrations of 1, 1.5 and 2 per cent with the V.I.C. and the V.O.C., with various fresh-gas flows. With the V.O.C. high vaporizer concentrations are needed, with the V.I.C. only low ones are necessary.

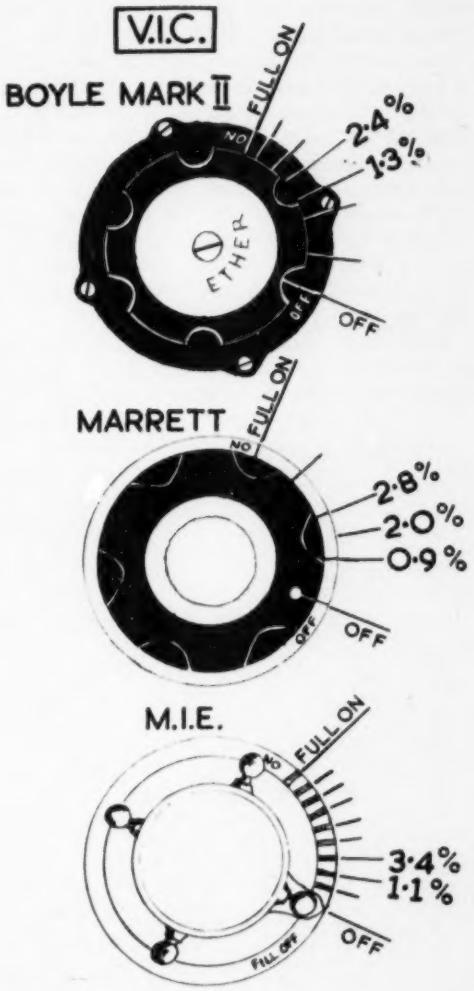

FIG. 5

Inspired concentrations of halothane measured in the operating theatre with three vaporizers used in the V.I.C. The M.I.E. tap shown here is the model prior to the recently introduced "Lincoln 800". Each vaporizer was used for a different patient; in all cases the fresh-gas flow was kept at 1000 ml/min, and the ventilation was 4-6 l./min. The angular movement of each tap to cover the clinical range of concentrations varied from tap to tap. For greater clarity, the direction of opening of the Marrett tap has been reversed.

volatile anaesthetic is obtained when a basal oxygen flow is used. With N_2O and O_2, the fresh-gas flow is unlikely to be below 1500 ml/min (vide supra). In any case the saving in volatile anaesthetic obtained by reducing the fresh-gas flow below 1000 ml/min is small enough to be neglected (fig. 1). With the V.I.C. the lower the fresh-gas flow the higher is the inspired concentration. Observations made in the operating theatre show that, for example, halving the fresh-gas flow in the circuit may increase the inspired concentration by as much as one-half (fig. 6).

With the V.O.C. the reverse effect is obtained. Lowering the fresh-gas flow lowers the inspired concentration (fig. 6). Figure 4 shows that if very small fresh-gas flows are used for reasons of economy, the vaporizer in the V.O.C. must be capable of delivering a concentration up to 6 per cent or more, if the inspired concentration is to be in the region of 2 per cent.

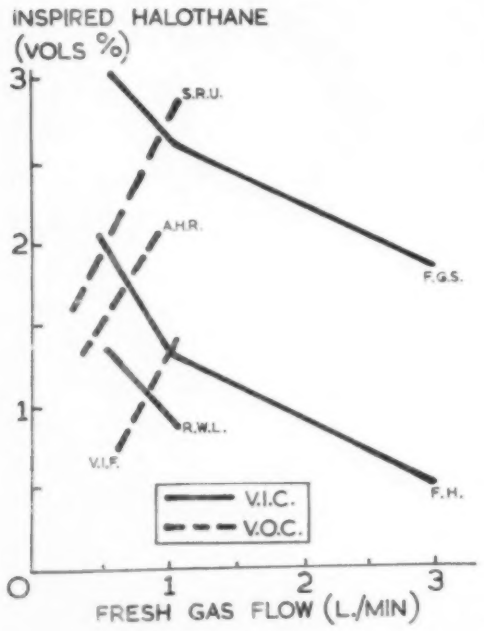

FIG. 6

The effect on the inspired concentration of halothane of changes in the fresh-gas flow in the V.I.C. and the V.O.C. The ventilations of the six patients were all within the range of 4–6 l./min. The tap setting was kept constant in each patient, though it varied from patient to patient. As the fresh-gas flow is increased the inspired concentration falls in the V.I.C. and rises in the V.O.C.

In general, therefore, with the V.O.C. low fresh-gas flows are indicated to give maximum economy and to keep the concentration of vapour in the circuit low. In fact, the only valid reasons for increasing the fresh-gas flow above basal in the V.O.C. are to compensate for leaks, to enable nitrous oxide to be used, and to compensate for a vaporizer of too low an efficiency.

In the V.I.C. on the other hand, caution must be exercised when trying to economize by using low fresh-gas flows. The vaporizer tap setting must be low when the fresh-gas flow is low, if too high a concentration in the circuit is to be avoided.

(c) *The minute-volume ventilation of the patient.*

With a patient breathing spontaneously, changes in the ventilation are outside the direct control of the anaesthetist. Whatever the position of the vaporizer in the breathing circuit, an increase in ventilation reduces the difference between the inspired and alveolar concentrations, and therefore tends to deepen the level of anaesthesia.

With the V.I.C., the ventilation is one of the most important factors affecting the inspired concentration. An increase in the ventilation leads to an increase in vaporization and therefore to an increase in both the inspired and the alveolar concentrations. Both theory and observation show that when the ventilation doubles, the alveolar concentration also doubles. This effect may be regarded as desirable, since an increase in spontaneous ventilation commonly results from stimuli during light anaesthesia; the increase in vaporization and hence in the inspired concentration will result in a deepening of the level of anaesthesia. There is, however, a real risk that a sudden bout of vigorous breathing may quickly produce high concentrations in the alveoli and blood which in the case of some anaesthetics may lead to serious cardiac effects before the respiratory centre is depressed sufficiently to reduce the ventilation. Conversely, if ventilation falls because of deepening anaesthesia, vaporization also falls off and

the concentration delivered to the patient is reduced. When spontaneous breathing is present, the dependence of inspired concentration on ventilation in the case of the V.I.C. tends to "smooth out" automatically the level of anaesthesia, and to act to some extent as a safety factor against accidental overdose.

However, the effect of ventilation on vaporization with the V.I.C. may be a disadvantage, if not a danger, when the anaesthetist assists or controls the ventilation. Unless he remembers to reduce the tap-setting or increase the fresh-gas flow, the increase in ventilation produced by squeezing the bag markedly increases the vaporization and therefore the inspired concentration. Good artificial ventilation of a deeply anaesthetized patient who would otherwise be breathing shallowly, nullifies the above "safety factor"; it is possible that overdosage due to this cause may have been responsible for some of the deaths reported with the use of halothane in the closed circuit (Chang et al., 1957; Foster, 1957; Payne, 1957).

With the V.O.C. a change in ventilation has no effect on the vaporization of the anaesthetic. An increase in ventilation produces a fall in the inspired concentration, and some rise in the alveolar concentration (Mapleson, 1960), thus tending to deepen the anaesthesia. (The exception to this is when a basal flow of oxygen is the only source of fresh gas; in this case the alveolar concentration is independent of ventilation.) The effect of ventilation on the inspired concentration is therefore opposite to that of the V.I.C. The magnitude of the effect on inspired and alveolar concentrations, of changes in ventilation with the V.O.C., is much less marked than with the V.I.C. (fig. 7), and depends on the fresh-gas flow and the level of ventilation. With fresh-gas flows of below 2000 ml/min and ventilations above 4 l./min there is very little effect on the alveolar concentration and hence on the level of anaesthesia (fig. 8). For example, in one patient with a fresh-gas flow of 2000 ml/min, a vaporizer concentration of 1.8 per cent, a ventilation of 5 l./min, and an alveolar concentration of 1 per cent halothane, an increase of ventilation to 10 l./min increases the alveolar concentration to 1.1 per cent, with negligible effect on the level of anaesthesia. However, it can be shown that, if the vaporizer concentration were increased to 6.3 per cent and the patient's ventilation fell to 2.5 l./min, his alveolar concentration would be 1.5 per cent. If now the ventilation were to be increased even to only 5 l./min, the alveolar concentration would rise to 3.3 per cent. Observations in the operating theatre have confirmed that changes in ventilation have very little effect on the depth of anaesthesia with the V.O.C., provided the ventilation is above 4 l./min.

This means that unless the ventilation is very low, assisted or controlled respiration can be instituted with greater safety with the V.O.C. than with the V.I.C.; on the other hand, with a patient breathing spontaneously, the V.O.C. does not carry the same "built-in" safety factor with respect to the effect of ventilation on inspired concentration that the V.I.C. possesses.

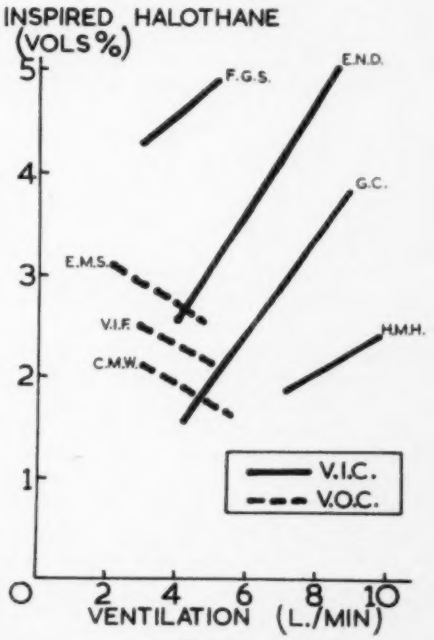

FIG. 7

The effect on the inspired concentration of halothane of changes in the minute-volume ventilation in the V.I.C. and the V.O.C. The fresh-gas flow was 1000 ml/min in all cases, and the tap setting was kept constant in each patient, though it varied from patient to patient. As the ventilation increases, the inspired concentration rises in the V.I.C. and falls in the V.O.C.

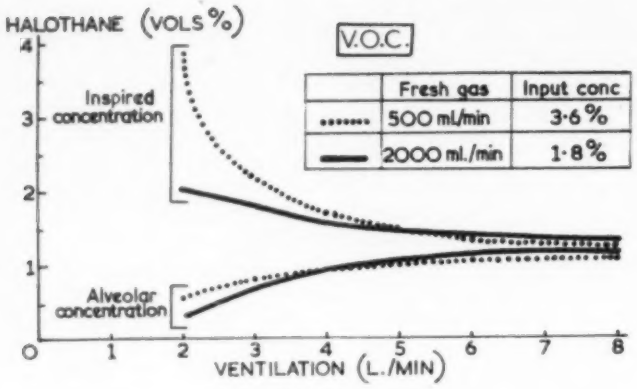

FIG. 8

A comparison of the effect in the V.O.C. of changes in ventilation on the inspired concentration and on the alveolar concentration. As ventilation increases, the difference between the inspired and the alveolar concentrations becomes less, but the alveolar concentration always rises with increasing ventilation and anaesthesia tends to deepen. These effects are greatest at low ventilations. As the fresh-gas flow is decreased, the effect of ventilation on the alveolar concentration decreases until at a basal flow the alveolar concentration is unaffected by changes in ventilation.

(d) *Uptake of anaesthetic.*

The rate of uptake of anaesthetic by the patient is the only factor that cannot easily be determined, but experiments still to be reported indicate that if the inspired concentration is constant, the rate of uptake of halothane falls rapidly at first but only very slowly after 20–30 minutes from the start of the administration. This has already been shown in animals (Duncan and Raventós, 1959), and similar reports with other anaesthetics indicate that the same is true for them (Haggard, 1924; Severinghaus, 1954).

It is clear that during an anaesthetic in which the parameters of tap setting, fresh-gas flow, and ventilation are constant, the uptake of anaesthetic into the blood from the lungs gradually decreases with time, as the body depots become increasingly saturated. It has been shown in the first paper in this series (Mapleson, 1960) that the effect of this is for the alveolar concentration, and hence the blood concentration, to rise gradually. In other words, the level of anaesthesia gradually deepens if the external factors are unchanged. This phenomenon is common to all inhaled anaesthetics, though the speed with which it takes place depends on the particular anaesthetic and on the fresh-gas flow. In bygone days this effect was referred to as Gill's "Law of Diminishing Resistance" (Gill, 1906), which, though originally explained in other terms, may nevertheless still be accepted as a guide to practice; the longer a patient is kept under an anaesthetic, the lower is the inspired concentration needed to maintain a steady level of anaesthesia. In the case of halothane the process is very slow and is of little significance unless the anaesthetic lasts more than 1–2 hours or is being administered to a child.

ESTIMATION OF INSPIRED CONCENTRATION

There are anaesthetists who believe that a knowledge of the inspired concentration may lead to a false sense of security, since they hold that the level of anaesthesia is dependent not only on the inspired concentration but also, more importantly, on the individual response of the patient to that concentration. Others, including the authors, are convinced that some knowledge of this concentration is necessary, if not essential, for the safer use in the closed circuit of anaesthetics as potent as halothane.

That an estimate of the inspired concentration

can be made with a fair degree of accuracy for a particular set of circumstances, in both types of closed circuit, is evident from the closeness between our observations made in the operating theatre, and the theoretical predictions.

With the V.I.C., changes in ventilation produce marked changes in the rate of vaporization, so that an estimate of the inspired concentration in the operating theatre may be rather complicated, though the graphs in this and the other papers in this series may be helpful.

With the V.O.C., however, the rate of vaporization remains constant if the fresh-gas flow through the vaporizer is constant. The fresh-gas flow is directly under the control of the anaesthetist. The uptake of anaesthetic by the patient can also be assumed to be reasonably constant after about 20 minutes. Unless the ventilation is very low, there will be little change in depth of anaesthesia as a result of changes in ventilation. A fairly close approximation of the inspired concentration can therefore be made in clinical circumstances with the V.O.C. by consulting the various graphs in this and the other papers. A more rough estimate is given by the following table, derived from observations made in the operating theatre.

Ventilation	Fresh-gas flow	Inspired concentration / Vaporizer concentration
4–6 l./min	300 ml/min	One-third
	500 ml/min	Two-fifths
	1000 ml/min	Three-fifths

It may be comforting to the anaesthetist to know that with the V.O.C. the inspired concentration will always be below that of the vaporizer concentration; the higher the fresh-gas flow, the nearer will the inspired concentration be to the vaporizer (or input) concentration.

"Build-up" of vapour concentration.

A source of anxiety to anaesthetists when using halothane in the closed circuit has been the fear that the inspired concentration will steadily go on rising because the circuit is "closed". In fact within 20 minutes, in any particular combination of circumstances (i.e. fresh-gas flow, ventilation and tap setting), the concentration rises to a level which then alters only very slowly, until the circumstances are altered. This does not mean, however, that dangerous or even lethal concentrations cannot occur. In any circuit, inappropriate combinations of tap setting, fresh-gas flow and patient's ventilation, can produce concentrations far above the clinical range. However, signs of overdose from too high a concentration are likely to show themselves within 20 minutes of any alteration in the combination of factors.

The explanation of this phenomenon has already been given in the accompanying papers. Briefly, an equilibrium is reached between the halothane added to the gases in the circuit and the halothane removed by the patient's body. Uptake of halothane by the body goes on steadily throughout even the longest anaesthetic.

A comparison of the V.I.C. and the V.O.C.

The following table sets out the advantages and disadvantages of placing the vaporizer inside or outside the breathing circuit.

VAPORIZER INSIDE THE BREATHING CIRCUIT (V.I.C.).

ADVANTAGES	DISADVANTAGES
(1) *Ventilation.* Apart from the tap setting this is by far the most important factor governing the rate of vaporization.	
1. If anaesthesia is too light, surgical stimulation will lead to an increase in ventilation and a deepening of anaesthesia.	1. A sudden increase in ventilation and therefore of the inspired concentration, may be dangerous.
2. If the tap setting is too high, deepening anaesthesia depresses the ventilation and reduces the vaporization; this acts to some extent as a built-in safety factor.	2. Increasing the ventilation by assisting or controlling the respiration without altering the tap setting leads to a marked increase in the inspired concentration.
3. If the patient stops breathing for any reason, no fresh vapour enters the circuit.	

(2) *Fresh-gas flow.* For any particular tap setting the smaller the fresh-gas flow the higher is the inspired concentration.

1. The smaller the fresh-gas flow, the greater the economy.	1. The smaller the fresh-gas flow, the higher the inspired concentrations. Economy is therefore obtained at the price of restricting the useful range of tap settings.
(3) *Vaporizer construction.*	
1. A simple low efficiency vaporizer is all that is required. There is little stimulus to provide a more elaborate vaporizer since ventilation is such an important factor affecting the inspired concentration.	1. The fact that the respirations pass through the vaporizer introduces problems of resistance.
(4) *Vaporizer concentration.*	
	1. Few calibrations have yet been made of vaporizers now in use.
	2. Even if the calibrations were known these would be of doubtful value because of the marked variation with ventilation. The inspired concentration is therefore difficulty to estimate.

VAPORIZER OUTSIDE THE BREATHING CIRCUIT (V.O.C.).

ADVANTAGES	DISADVANTAGES
(1) *Ventilation.* This has no effect on the rate of vaporization.	
1. Assisted or controlled respiration has little effect on the depth of anaesthesia and therefore is safer than with the V.I.C.	1. An increase in ventilation due to surgical stimulation in light anaesthesia does not produce the automatic compensation of increased vaporizaton that occurs with the V.I.C.
	2. Conversely, too deep anaesthesia with respiratory depression does not have the "built-in" safety factor of the V.I.C.
(2) *Fresh-gas flow.* For any particular tap setting* the smaller the fresh-gas flow, the lower is the inspired concentration.	
1. The smaller fresh-gas flows provide both greater economy and lower inspired concentrations. Economy does not introduce the risk of overdose.	
(3) *Vaporizer construction.*	
1. Thermostatically controlled vaporizers are available.	1. Thermostatic vaporizers are costly.
(4) *Vaporizer concentration.*	
1. Calibrated vaporizers are readily available.	
2. An estimate of the inspired concentration can be made with fair accuracy and little difficulty.	

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* *N.B.* In the case of the Fluotec vaporizer, although a reduction in fresh-gas flow, particularly below 1000 ml/min, leads to an increase in vaporizer concentration above that indicated by the tap, the above effect still predominates.

CONCLUSION

It is apparent that each circuit has its own advantages and disadvantages. It must be left to the individual anaesthetist to decide which is better suited to the particular circumstances of his own clinical practice. A minimum requirement, however, is that he should understand clearly the way in which the alveolar concentration and hence the depth of anaesthesia is dependent on the factors of ventilation, fresh-gas flow, and vaporizer characteristics.

It seems to us that in the hands of an experienced anaesthetist either arrangement is equally safe (or unsafe). If the anaesthetist is less experienced with potent anaesthetics such as halothane, he is better advised to use a calibrated and preferably thermostatically controlled vaporizer placed outside the breathing circuit, especially if he intends to assist or control the respiration. By this means the somewhat unpredictable effects in the V.I.C. of alterations in the patient's ventilation on the inspired concentration of anaesthetic are avoided, particularly if controlled respiration is performed.

ACKNOWLEDGMENTS

We thank our colleague, Dr. W. W. Mapleson, Ph.D., Physicist in this Department, for his freely given help. Without his theoretical work our paper would have lacked foundation. He has generously allowed us to use much data from his theoretical studies in drawing many of our graphs and given valuable literary and scientific criticism in aid of clarity and accuracy.

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UNINTENTIONAL SPREAD OF EPIDURAL ANALGESIA

BY

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AT a meeting recently the topic for discussion was: "Is epidural analgesia worth the bother?" One might as well ask if the choice of ether or cyclopropane is ever justified, because, like epidural analgesia, an irrefutable incidence of spectacular complications accompanies their employment—explosions with ether and cyclopropane and total spinal blocks with epidural analgesia. It is nevertheless a fact that epidural analgesia is altogether eschewed for that reason in many reputable centres the world over.

If epidural analgesia is ever to attain its rightful place in anaesthesia, then all the possible mechanisms for its complications must be clearly recognized.

It is today not enough merely to avoid inadvertent subarachnoid or intravenous injections, because those account only for immediate reactions, whereas the delayed complications are becoming increasingly recognized. They may occur unexpectedly some 15 to 40 minutes after the injection is completed (Morrow, 1959; Stovner, 1957; Sykes, 1958) or they may only become apparent postoperatively as persistent neurological defects (Wolfson and Ingram, 1957; Davies et al., 1958).

The mechanism of all these delayed sequelae is still shrouded in mystery.

This paper attempts to relate the thorough work of Moore et al. (1954) on the intraneural spread of fluids in monkeys to the occurrence of delayed complications of epidural analgesia.

METHODS

On many occasions a large nerve of an extremity was dissected out immediately after amputation in the operating theatre. It was gratifying to be able to demonstrate how easily intraneural spread occurred and often how little pressure was required to accomplish this. Moore et al. (1954) state that it is, on occasion, as easy as intravenous injection. Thus, cross sections of the nerve 2 to 3 inches from the site of an injection of 2 per cent lignocaine containing 1 per cent methylene blue displayed clearly the nerve fasciculi macroscopically visible in a sea of blue (methylene blue occupying the perineural spaces).

A brief apologia is required before describing another experiment. Moore et al. (1954) demonstrated in monkeys that solutions coloured with methylene blue and injected 3 to 4 cm lateral to the intervertebral foramina, under direct vision, reached the parenchyma of the spinal cord in 2 to 5 minutes. The spinal fluid did not immediately become tinged with the dye, but it required 10 to 15 minutes for the solution to pass through the pia mater and epineurium to stain the spinal fluid lightly, and it did not become heavily stained for 35 to 40 minutes. Morrow (1959) has probably reported the longest delay before the onset of total spinal block after intended epidural injection, namely 40 minutes. Unfortunately these workers used Efocaine, a known neurogenic poison (Scott, 1958) which caused many mishaps after paravertebral block during the first few years of the last decade. Transverse myelitis, Brown-Séquard syndrome, neuritis, inflammatory reactions and lasting absence of nerve function were all attributed to the action of Efocaine (Nowill et al., 1953a; Nowill et al., 1953b; Moore, 1954; Moore et al., 1954; Parsonage et al., 1955). A severe destructive effect of the major solvent vehicle, propylene glycol, was demonstrated on rabbit tissues in 0.5 ml doses, even in a dilution as great as 1:8 (Margolis et al., 1953). These experiments of Moore et al. (1954) nevertheless demonstrated that a substance can be introduced mechanically into the cerebrospinal fluid and spinal cord by direct injection into the nerves distal to the intervertebral foramina, quite apart

from any injection into an outward prolongation of the subarachnoid space. There is no reason or evidence to suggest that Efocaine is unique in this respect, especially in view of the relatively large size of the dorso-lumbar nerve roots in the human being, and their transfixion in the intervertebral foramina—as opposed to the cervical roots of the brachial plexus.

In order to clarify this point and to exclude the confusing neurotoxic properties of Efocaine a large dog was operated on under halothane anaesthesia. A lumbar nerve root was mobilized and using a No. 18 hypodermic needle, carefully injected directly at a site 1 inch (2.5 cm) from the spinal cord. About 5 ml of 2 per cent lignocaine, containing 1 in 200,000 adrenaline and 1 per cent methylene blue were injected at a rate of 1 ml every 30 seconds.

Within 15 seconds blue streaks appeared on the exposed part of the spinal cord, and they gradually became thicker and more numerous until, after 60 to 75 seconds, the whole of the exposed portion of the dura mater suddenly turned blue (figs. 1 and 2).

Up to this time the depth of anaesthesia was maintained at a plane which allowed movements of the limbs to occur almost without interruption. Although the general anaesthetic was now completely withdrawn, the dog only managed to move his head. The paraplegic state was allowed to persist for half-an-hour, when the dog was sacrificed with an overdose of halothane and the whole spinal cord dissected out; it was found to be stained blue along its whole length, more intensely in the lower dorso-lumbar area, as was to be expected.

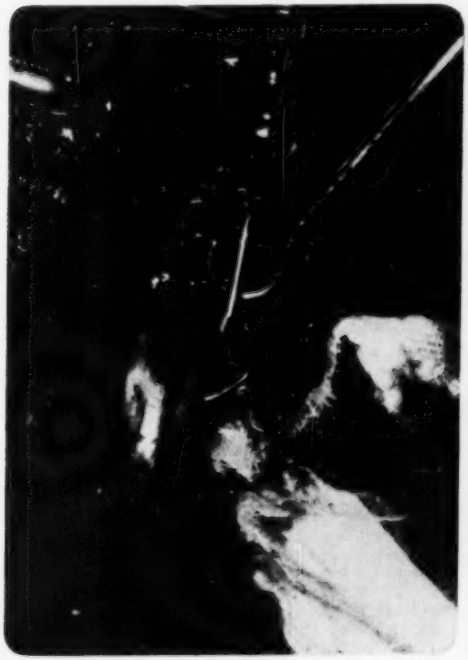

FIG. 1

A lumbar nerve in a dog being directly injected with a lignocaine-methylene blue solution; showing the cephalad spread into the spinal cord. See also figure 2.

FIG. 2

Same dissection as in figure 1, but a minute later at the exact moment of rupture of epineurium and pia with instantaneous discoloration of the whole spinal cord.

DISCUSSION

The proof of a spread centrally via the perineural spaces of lignocaine in figures 1 and 2, is perhaps clearer, more striking and convincing than the beautiful illustration of the same avenue of spread of Efocaine on page 52 of Moore's (1955) book on the complications of regional blocks. This is exactly what a consideration of the anatomy will demand, because a nerve root consists of fasciculi, each surrounded by perineurium and perineural spaces, the whole root being surrounded by the epineurium. After intraneural injection, spread therefore occurs initially along the perineural spaces only and may continue thus directly into the spinal cord (fig. 1). With mounting pressure the relatively fragile epineurium, and especially its continuation the pia mater, are stretched, the latter thus rendered more permeable to solutions (Moore et al., 1954) until the pia mater may suddenly rupture with wide subarachnoid dissemination of the injected solutions as a result (fig. 2).

Clinically a patient has been noted in whom a neurological lesion, consisting of lower limb motor defects coupled with preservation of more superficially situated sensory tract function, persisted after spinal analgesia was induced during thiopentone anaesthesia (Mostert, 1958). Surely intraneural or even direct injection into the spinal cord must be invoked as a possible, or even probable cause. Although in 94 per cent of humans the spinal cord terminates in the region of the first lumbar vertebra, in some it may end as low as the third. Dripps and Vandam (1951) encountered paraesthesia in 13 per cent of a series of lumbar punctures.

While intraneural injection must be very rare during the induction of epidural analgesia, there has been a failure to recognize it as a prelude to disaster by all the British authors referred to in this article and this has led to a lack of faith in the utility of the test dose and a lack of appreciation of the need for good rapport with the patient during the epidural injection in order to avoid pain and paraesthesia, and so intraneural injection. This is particularly unfortunate, because the fact that no spinal fluid is obtained, even on aspiration, is no assurance that the needle is not in the intrathecal space (Adriani et al., 1952). Two cases of total spinal block were reported by Bonica et al. (1957) despite the presence of this sign, but a further 17 total blocks were inevitable in their series of 3,637 cases had it not been for their routine use of the test dose.* In any case, it is faulty reasoning (similar to stating the conclusions of an experiment before it has taken place) to say that a test dose followed by a 5 minutes waiting period is without value becauses widespread subarachnoid extension may be delayed up to 40 minutes. Finally it is essential, when investigating these complications, to know whether pain, paraesthesia, and weakness were present after the test dose, as well as knowing whether spinal fluid or blood was encountered prior to the block.

SUMMARY AND CONCLUSION

Previous work on the intraneural spread of Efocaine in monkeys is reconsidered in the light of a similar sequence demonstrated in a dog injected with lignocaine, and in human nerves obtained from amputated limbs. The conclusion, shown to be entirely feasible, is that a spread via the perineural spaces must be considered in any attempt to account for the occurrence of unexpectedly delayed extension of epidural analgesia. To avoid these complications good rapport with the patient during epidural injection in order to avoid all pain and paraesthesia and the routine use of a test dose are absolute requisites.

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* During the past year the author personally administered or supervised 500 administrations of epidural analgesia between thoracic 8 and lumbar 5 vertebrae, at the King Edward VIII Hospital in Durban. The conscious patients were invariably sitting up; their ages varied between 7 and 90. An ordinary 10 cm 18 S.W.G. autoclaved needle provided as a routine for drawing up of drugs was used, and the analgesic solution consisted of 8 to 22 ml 2 per cent lignocaine with adrenaline. *A test dose was not used*, but all other precautions were observed. Three total spinal blocks, complete with respiratory arrest during the hypotensive period, irregular pupils for the duration of unconsciousness, and tolerance of an endotracheal airway in the presence of good pharyngeal tone *unexpectedly* ensued after two to *twenty-two* minutes the earliest total block progressing to cardiac arrest. A fourth patient had convulsions remedied with suxamethonium, and a fifth brief but profound hypotension after her legs were lowered from the lithotomy position, both prior to Caesarean section. There were nine failures, no neurological sequelae, and all recovered completely (100 Caesarean sections were in the series), but today all the participating colleagues agree on the grave necessity of the test dose.

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BOOK REVIEW

A Symposium on pH and Blood Gas Measurement: Methods and Interpretation. Edited by Ronald F. Woolmer, assisted by Joy Parkinson. Published by J. & A. Churchill Ltd., London (1959). Pp. 210; illustrated. Price 30*s*.

This is likely to be a "Bible" for research workers in this field for some time to come. A great deal of experimental work in anaesthesia has been based upon pH estimations which by modern standards would be quite unacceptable. Professor Woolmer did a great service by arranging the Symposium, the proceedings of which are here reported, and by persuading undoubted international authorities to contribute. We can only endorse the remarks of Professor Woolmer in his Foreword: "that this book brings together a wealth of information about a subject so diffuse that no textbook deals with it as an entity; and though it does not set out to cover every aspect fully, there are not many which escape mention in the discussion or in the references, even if they are not dealt with in one of the ten papers". The Symposium was held in three sessions. Each paper was followed by a discussion and there was a general discussion on the whole subject of each session. All of this is here reported and as is usual in this type of work the discussions are most instructive material.

Among the most valuable subjects covered are the interpolation methods of estimating blood P_{CO_2}; the accuracy of which is fully evaluated; the problem of whether "true" and "separated" plasma are identical for the purpose of equilibration techniques; the value of the end-tidal sample as an estimation of arterial P_{CO_2}; and particularly useful are the sections during the first and third sessions devoted to a consideration of the various electrodes for pH measurement, the direct estimation of blood O_2 and CO_2 and the electrochemical aspects of blood pH measurement. This book has already become a standard reference. *Cecil Gray*

Brit. J. Anaesth. (1960), **32**, 338

A TRUE CIRCLE CONVERSION OF THE "C-M" ABSORBER UNIT

BY

T. A. BROWN AND M. A. WOODS

North Down Group of Hospitals, Northern Ireland

THE advent of halothane has led to some revival of interest in inhalation anaesthesia with spontaneous respiration, muscular relaxation being obtained by deepening the plane of narcosis rather than by the use of specifically relaxant drugs. In cases so conducted significant alveolar accumulation of carbon dioxide must follow the inevitable respiratory depression. This is especially so when a completely closed circuit technique is used—probably due to the absence of the flushing effect produced in the respiratory tract by the high gas-flow normally employed in semi-closed systems.

A series of more than 5,000 patients having closed circuit halothane anaesthesia was closely observed. The inclusion of the halothane vaporizer in the circuit precluded assisted respiration and minute volumes of 3 litres and less were not uncommonly encountered. No untoward cardiovascular effects were observed. Nevertheless, respiratory acidosis during anaesthesia can never be regarded as beneficial and every effort should be made to ensure that, in closed-circuit systems, the atmosphere inhaled by the patient contains as little carbon dioxide as possible. This is achieved by using an efficient system of absorption.

Probably the most common closed-circuit machines in this country are the "C-M" units, marks 1 and 2. About 1,750 of these were distributed in the British Isles before the design was superseded by the Boyle II circle absorber (Bracken, 1960). The units are robust, convenient and efficient in all but one respect. Due to the method used to embody the to-and-fro principle of carbon dioxide absorption, a deadspace of about 160 ml occurs above the soda lime canister (Bracken, 1960). At the end of each expiration, this space contains mainly exhaled gases untouched by soda lime and therefore rich in carbon dioxide. Inspiration draws towards the lungs a mixture of this unaltered exhalation and gases from the rebreathing bag which have passed twice through the soda lime. Some carbon dioxide is therefore re-inhaled.

To get an approximate estimate of the extent of this re-inhalation, a conscious volunteer breathed from a "C-M" unit. Basal oxygen was supplied and the freshly filled absorber was turned full on. A carbon dioxide analyzer working on the colorimetric principle was interposed between the unit and the inspiratory hose. At the end of 5 minutes quiet respiration the analyzer showed that the inspired atmosphere contained 3 per cent carbon dioxide. During the respiratory depression of deep halothane anaesthesia, tidal volume may fall to 100 ml or less. In such circumstances the deadspace in the "C-M" unit furnishes a large proportion of each inspiration, and the level of inspired carbon dioxide exceeds 5 per cent, which is the highest reading possible with the colorimetric analyzer used.

Therefore, although laboratory experiments with an artificial lung show an advantage in absorptive efficiency from the use of the to-and-fro method (Adriani and Rovenstine, 1941), it is technically difficult to reproduce this advantage under working conditions with the "C-M" absorber as originally designed.

It must be accepted that a true circle system, which can contain no deadspace, is more efficient during anaesthesia.

Any "C-M" unit can be easily converted to the true circle principle by slightly altering the relative positions of its components as follows. The soda lime canister is removed from its usual position. The tapered stub at its upper end is plugged into the socket in the unit which normally receives the mount of the patient's expiratory hose. The screwcap at the bottom of the canister is modified as described and illustrated below, and the expiratory hose mount plugged

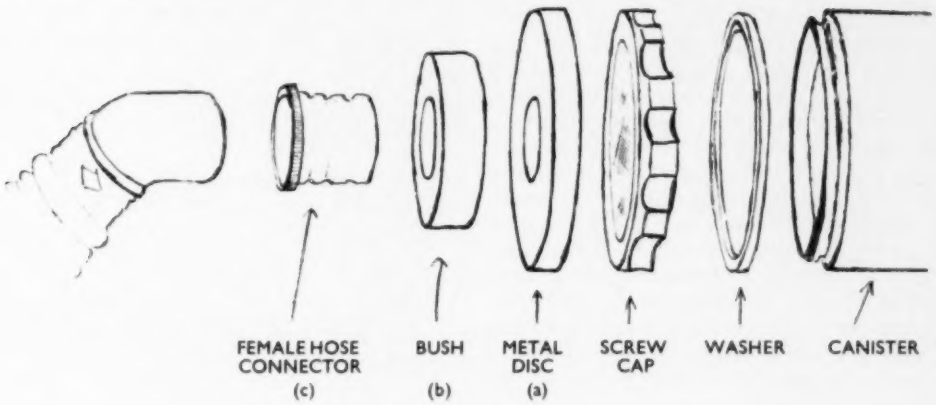

FIG. 1

into it. The absorber control drum on the unit is turned permanently to the mark "Off" to avoid gross leakage of the circulating gases.

In use, all the patient's expirations must now pass through the absorber canister on their way to the rebreathing bag, and a unidirectional flow of the circulating gases is ensured.

Repetition of the previously mentioned experiment with a conscious volunteer breathing from the converted unit demonstrated a considerable gain in absorptive efficiency. No carbon dioxide could be detected in the inspired atmosphere after 5 minutes, showing the inhaled percentage of this gas to be less than 0.3 per cent (the lowest concentration measurable with the analyzer used).

Similar investigations during deep closed circuit anaesthesia with halothane failed to detect any carbon dioxide in the inspired atmosphere even at the lowest minute volumes.

MECHANICAL DETAILS

A circular disc of copper or brass about $\frac{1}{4}$ inch thick and $2\frac{7}{8}$ inches in diameter (fig. 1a) is reinforced by brazing to one side of it a brass nut or bush about $\frac{1}{2}$ inch thick and 2 inches in diameter (fig. 1b). A hole is now reamed through the centre of this assembly to fit snugly a standard female hose connector (fig. 1c) which is brazed into position with its small end flush with the plain side of the brass disc. The plain side of the disc is then sweated on to the outside of the screw cap of the soda lime canister and a ring washer is cut from resilient plastic sheet to fit closely over the threads of the screw cap. This must be thick enough to give a gastight joint when the cap is screwed on to the canister.

As the canister is used in the horizontal position, incomplete filling with soda lime will lead to the exhaled gases taking the line of least resistance by passing over the top of the soda lime rather than through it. A domestic nylon pot scrubber compressed between the screw cap of the canister and the soda lime readily solves this difficulty and causes little added resistance to respiration (Robson and Pask, 1954).

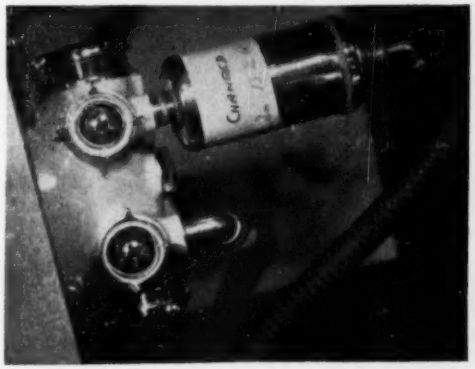

FIG. 2
Showing modified canister in position.

SUMMARY

The design of the "C-M" absorber is such that there is an appreciable deadspace (circa 160 ml) at the mouth of the soda lime canister. Experiments are described which show that, in anaesthesia with quiet respiration, this deadspace may lead to a serious increase in carbon dioxide in the inhaled atmosphere, even exceeding 5 per cent in certain circumstances.

A simple modification of the unit is described. By altering the position of the soda lime canister, so that it becomes monophasic instead of biphasic, the deadspace is removed and carbon dioxide accumulation is prevented.

ACKNOWLEDGMENTS

We are greatly indebted to Dr. A. Bracken of British Oxygen Research and Development Ltd., for information relating to the Coxeter-Mushin absorber units; to J. MacGouran, M.S.R., for the photograph, and to Mr. M. Stevenson of our hospital engineering staff who undertook the modifications to the canisters.

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FACULTY OF ANAESTHETISTS OF THE ROYAL COLLEGE OF SURGEONS IN IRELAND

At a meeting held in the Royal College of Surgeons in Ireland on Friday, July 8, 1960, the President of the College, Mr. T. G. Wilson, inaugurated a new Faculty of Anaesthetists. The first Board of the Faculty consists of:

Dr. Thomas James Gilmartin, *Dean*
Dr. Victor Ormsby McCormick, *Vice-Dean*
Dr. Joseph Augustine Woodcock, *Hon. Sec.*
Dr. Patrick Joseph Drury Byrne
Dr. Patrick Joseph Nagle
Dr. Ethel Sheila Kenny
Dr. Paul Finbar Murray
Dr. Geoffrey Raymond Davys
Dr. John Wharry Dundee
Dr. Samuel Harold Swan Love

After admitting the Board members as Foundation Fellows in the Faculty, the President also admitted the following Foundation Fellows:

Doctors Sachindra Nath Basu, Kathleen Bayne, William Bingham, John Boyd, George John Cecil Brittain, Daniel Gerard Coleman, Walter Samuel Davis, Kevin Bernard Glynn, Thomas Francis Heavey, John Cecil Hewitt, Margaret Marion Vida Lemon, John Alexander Macaulay, Mary Josephine Ahern O'Connor, Margaret Silver Deane Oliver, Lady Frances Edna Read, Richard William Shaw, and Maurice Alexander Woods.

The following Foundation Fellows were also conferred in absentia:

Doctors James Daniel Bourke, Arthur Barclay Bull, Francis MacDermot Byrn, Anthony Halton, Kasasian, Ordio Victor Steyn Kok, Zoltan Lett, Brendan Vincent Lyne, Paul Ronald Mesham, Maureen Murphy, and David Lindsay Scott.

The Honorary Fellowship in the Faculty was then conferred by the President on Dr. John Gillies, who was introduced by the Vice-Dean, Dr. McCormick, and upon Dr. Geoffrey Stephen William Organe, who was introduced by Dr. Drury Byrne.

CORRESPONDENCE

NEONATAL PHYSIOLOGY AND RESUSCITATION

Sir,—Dr. Selwyn Crawford in his letter on "Neonatal Physiology and Resuscitation" (*Brit. J. Anaesth.* (1960), **32**, 295) has performed the signal service of making me re-examine my notions as to blood volume and umbilical vein oxygenation in the small baby as expressed in my article "Infant Physiology and Anaesthesia" (*Brit. J. Anaesth.* (1960), **32**, 22). I think he must realize, nevertheless, that that paper put forward principles of infant physiology which were of practical value to paediatric anaesthetists, and certainly did not pretend to give an exhaustive coverage of neonatal blood volumes or gas tensions.

Accurate determinations of these values are notoriously difficult to obtain, but I still accept the figures from Walker (1954) of Aberdeen who showed that foetal umbilical vein oxygen saturation fell from about 70 per cent at 30 weeks to about 60 per cent at 39 weeks, and then to about 30 per cent at 43 weeks. Considering that umbilical vein blood is mixed with still less saturated blood from the foetal systemic circulation I can only repeat my statement that "the foetus is accustomed to comparatively poor oxygenation at term." Living on the inferior surface of the globe I have not yet received the symposium quoted by Dr. Crawford but I am in some doubt as to whether it will make me change my views. Our babies, anyway, are born of a hue which approximates more to the right than the left hand end of the spectrum.

In regard to blood volumes I think it is very interesting to read the excellent articles by Sisson et al. in the *Journal of Pediatrics* 1959 and 1960. If Dr. Crawford were to re-read the first of these, he would see that the mean blood volume of the baby on the first day was nearly 100 ml/kg, rising by the sixth day to 110 ml/kg. This I confess is less than my suggested "nearly 120 ml/kg", but slightly closer to the latter than to his 85 ml/kg. After some fluctuations the blood volume at five months averaged 95 ml/kg and by twelve months was 85 ml/kg. (My average figure was 90 ml/kg.) All these values are subject to considerable individual variation, but my intention in quoting the figures I did, was to give a reasonable basis on which to manage the infant's blood and fluid replacement during the period under discussion.

It was a little unfair of Dr. Crawford to suggest that I recommended (with Professor Donald) the use of gastric oxygenation rather than the passage of an endotracheal catheter and the use of I.P.P.R. The former method is used more often than the latter because not many people other than paediatric anaesthetists have much practice at passing endotracheal tubes on newborn babies. The unpractised person is much more liable to do harm than good, whilst if gastric aspiration is practised as a routine (as it is at the Royal Women's Hospital, Melbourne) it involves little effort to establish gastric oxygenation. There are some obstetricians and obstetrical anaesthetists who maintain that such a method is useful. But by all means let the experienced operator use endotracheal I.P.P.R. with oxygen if the indications warrant it.

IAN H. MCDONALD,
Royal Children's Hospital, Melbourne, Australia.

Sir,—Perhaps Dr. Crawford would be good enough to read my article again before he next puts pen to paper. It is quite clear from this article that endotracheal intubation takes pride of place in meeting respiratory emergencies at birth and it is agreed that gastric oxygenation is less effective. My words were: "A simple though far less effective alternative is to administer gastric oxygen." This latter method is so widely practised and, even though ineffective, is relatively so harmless that a majority of clinicians would have deplored its omission from the article and Dr. Crawford is in too small a minority to cater for. He seems to think that even oxygen administered by a funnel pressed over the mouth a futile procedure. From the practical point of view

even a poor method is better than no method at all and I would remind Dr. Crawford of the regrettable fact that by far the majority of births in this country take place in the absence of the necessary skill in intubation on the part of those present; so, let us at least face practical facts.

Woolly reference should not be given to unpublished work or work which cannot be quoted properly. Dr. Crawford may be interested to note an article by Coxon (1960), *Lancet*, **1**, 1315.

The terms "blue asphyxia" and "white asphyxia" with which I am "chided" are in such common clinical usage that again they cannot be ignored, but if Dr. Crawford will look again I think he will see that I have said, "it would be better to describe this condition (white asphyxia) simply as foetal shock rather than asphyxia pallida since the former term gives a far better picture of what is happening".

The Apgar scoring system has already been referred to by me in my textbook *Practical Obstetric Problems*, 2nd edition, page 441, which your correspondent may care to look up. It is certainly a good system provided staffing is adequate, which is seldom the case.

IAN DONALD

Western Infirmary, Glasgow

THE FIRST ADMINISTRATION OF CHLOROFORM

Sir,—With reference to Dr. Langton Hewer's letter in the *British Journal of Anaesthesia*, for May 1960 (page 234), I should like to make the following points. I think it is by no means certain that chloroform was in fact used as early as the spring of 1847. The evidence in support of Dr. Holmes Coote's precedence is based on a letter written 24 years after the event. It used the expression "chloric ether (which is chloroform plus spirits of wine)". I am not aware that a solution of chloroform in spirits of wine was ever called "chloric ether"; I suggest that Mr. M. C. Furnell, who wrote the letter, may well have been mistaken. As I pointed out in the article which Dr. Hewer criticizes, "chloric ether was also used in the Middlesex Hospital as early as February 1847". While, of course, Dr. Holmes Coote's priority may be a fact, it is my belief that more satisfactory, contemporary evidence is required to prove it.

M. H. ARMSTRONG DAVISON

Department of Anaesthetics,
The Medical School,
Newcastle upon Tyne, 1

THE FIRST ENDOTRACHEAL INTUBATION

Sir,—In his interesting article, "The Evolution of Endotracheal and Endobronchial Intubation", Dr. White states that Benjamin Pugh was the first to perform intubation. I take leave to doubt this. As I pointed out in my article in the *British Journal of Anaesthesia*, 1956 (page 577), Benjamin Pugh undoubtedly described an air-pipe for the resuscitation of the newly born; It was made of a wire spring, 10 inches long, covered with thin soft leather, and was to be introduced into the infant's mouth "as far as the larynx". Even if these last words be discounted, I think it is extremely unlikely that, at that date, a wire spring covered with leather could have been made sufficiently small to be inserted into the larynx of a neonate. In the absence of further evidence, I am not prepared to accept Benjamin Pugh as the originator of intubation, and I believe that the honour for this rests with Charles Kite (1788), acting on the suggestion of "Dr. Munro, Mr. Portal, Mr. le Cat and others".

M. H. ARMSTRONG DAVISON

Department of Anaesthetics,
The Medical School,
Newcastle upon Tyne, 1

AN ANAESTHETIC HAZARD

Sir,—Robbie and Pearce (1959) drew attention to some dangers associated with latex armoured endotracheal tubes. In the second case they reported, a layer of latex rubber had puckered up inside the tube and occluded the lumen.

I have to report a similar occurrence in conducting rubber non-kinkable catheter mount tubing. The tubing which had been in use for some months was connected to a Magill T-piece. Total obstruction of the airway was immediately apparent and was tracked down to the catheter

mount. A layer of the antistatic rubber had become detached by the introduction of the T-piece and had puckered up, causing complete occlusion of the lumen (fig. 1b). No undue force was required to insert the T-piece.

Although the cause of the obstruction was apparent on examining the catheter mount, it was possible for the rubber lamina to spring back into its normal position on disconnecting the T-piece (fig. 1a).

It would seem that the introduction of metal coiling into rubber, whether latex or antistatic, introduces the risk of layering and, unless a more satisfactory binding of the rubber can be achieved, it might be safer to use a short length of ordinary antistatic rubber of suitable bore and rigidity.

A. H. B. Masson
The Royal Infirmary, Edinburgh

REFERENCE
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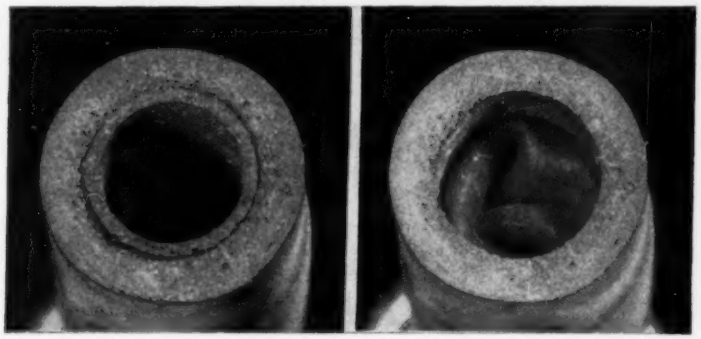

(*b*) (*a*)

Fig. 1

Magnified view of distal end of armoured catheter mount tubing:
(*a*) Showing separation of inner lamina;
(*b*) Showing obstruction of lumen.

BOOK REVIEW

A Practice of General Anaesthesia for Neurosurgery. By Robert I. W. Ballantine, M.R.C.S., L.R.C.P., D.A., F.F.A.R.C.S., and Ian Jackson, M.R.C.S., L.R.C.P., D.A. Published by J. & A. Churchill Ltd., London. Pp. viii + 152; 68 illustrations. Price 27*s*. 6*d*.

How right Mr. O'Connell is in his foreword to the book, in which he states that "In the past, neurosurgical anaesthesia has appeared unattractive to some and was avoided for what was considered to be more rewarding work."

The authors have not set out to produce a textbook of neurosurgical anaesthesia, dealing with all possible techniques, but "to describe in detail general anaesthetic techniques that we have found simple and efficient in neurosurgery". By doing so they have filled a great gap in anaesthetic literature and, while there are occasions where those engaged in neurosurgical anaesthesia may question the wisdom of their techniques, they do offer a presentation which will benefit any trainee anaesthetist who reads the book.

Their first two chapters on general considerations, particularly dealing with those problems due to intracranial pressure, are of great value and are worthy of committing to memory. They have an excellent section on venous pressure and issue a warning that "It is not difficult to appreciate how a patient with a high intracranial pressure may be brought to the verge of immediate death . . . by coughing during induction."

Their advice on premedication in Chapter 3 is very sound, particularly as regards heavy sedation, but no mention is made of the cases who received such sedation in general surgical or medical wards before being handed over to the care of the neurosurgical anaesthetist. In this same chapter, one would have thought that there might have been a place for mentioning Anderson's scheme for opiate premedication in children and also for some of the newer pediatric techniques.

It is to be noted, in the chapter on techniques, that the authors have by no means abandoned the use of trichloroethylene for the much more expensive and often less satisfactory halothane, and one hopes that this advice will be taken to heart by the younger anaesthetists. Their routine e.c.g. monitoring on cardiac rhythm must set at ease the minds of those who routinely combine adrenaline with trichloroethylene. On page 35 one wonders whether or not they can have shown correlation between hypercarbia and ventricular irregularities with this drug.

The authors have been very frank in their views concerning controlled respiration and hypothermia and, while they themselves do not apply these routinely, they have by no means condemned them and have presented a very fair picture of their merits and demerits.

Those who have had no experience of neurosurgical anaesthesia may find Chapters 7 and 8, dealing with individual operations, somewhat confusing, but this is experience which can only be gained in the operating theatres and, for such, this book provides an excellent background. One notable mention is of the devotion of only three lines to the prone position for occipital operations (page 102) despite previous mention of the dangers of abdominal pressure in this position (page 15).

This book is very well set out, on first-class paper, and contains 68 illustrations. The showpiece is, without doubt, the record cards of craniopagus twins (pages 44 and 45). However, one wonders if 27*s*. 6*d*. is not a little bit too much for a book of this nature. Many of the illustrations are record charts from actual operations, and at least 15 of these could be left out without detracting from the value of the book and probably reducing it considerably in price. In many instances two records could be included in one page by omitting the right half of the standard Nosworthy card.

One may also criticize the use of milligrams instead of the more useful milli-equivalent throughout the book. On page 20 we have the most peculiar formula for quinalbarbitone which, although correct, does not conform to the standard terminology used in this country. These are small details which should not detract too much from the value of this book as a useful contribution to the anaesthetic literature.

John W. Dundee

Printed in Great Britain by John Sherratt & Son, Park Road, Altrincham

trophenium

REGD. phenactropinium chloride

an agent for the production of controlled hypotension during general anaesthesia, providing ganglion blockade without direct vasodilator action. This original product of the Duncan Flockhart Research Laboratories is now available in the new, more convenient, pack of 20 ml. (1,000 mg.) in a box of 6 injection-type vials.

References: "The use of a homatropinium derivative to produce controlled hypotension" *Brit. J. Anaesth.* **29**, 342.

"Comparison of two hypotensive agents" *Anaesthesia*, **14**, 53.

"The use of Fluothane and Trophenium in anaesthesia for the surgery of deafness" *Scot. med. J.*, **3**, 496.

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